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The
Medical Clinics of Chicago

VOLUME 1 NUMBER 6

CLINIC OF DR. WALTER W. HAMBURGER

COOK COUNTY HOSPITAL

THE ALLEN TREATMENT OF DIABETES

Fundamental Principles. Treatment Divided into Three Stages: Prolonged Starvation, Tolerance Testing, After-care.

Results in 14 Patients; Representative Cases; Methods of Treatment. Treatment of Severe Cases. Modification of Allen Method; Daily Progressive Dietary with Specimen Diet Charts Arranged by Miss Maude E. Perry, Dietitian to the Michael Reese Hospital.

Advisability of Keeping Patients Sugar-free at Sacrifice of General Well-being.

Ultimate Success of Treatment Depends on Extensive Observation and Experience.

AT the meeting of the Association of American Physicians at Washington last May Dr. Frederick M. Allen presented a report of his work on prolonged fasting in diabetes. It was my privilege to be present and to hear the discussion of Dr. Allen's paper. Deeply impressed with the results obtained at the Rockefeller Hospital in 44 cases of severe and moderately severe diabetes, I resolved to apply this method in the treatment of diabetes at the County Hospital. Since that time, that is, during the past nine months, we have had the opportunity of studying 14 cases of diabetes under prolonged fasting. I should like to present to you today the results of these cases, together with the impressions and experiences received during their treatment. In addition, I should like to discuss a rather schematic

emphasized. During this period, as Joslin[1] particularly has pointed out, one should guard especially against the reappearance of glycosuria and acidosis (from an improper diet) because of the danger of such relapse in a patient who has lost considerably in weight, and who has already become somewhat weak and exhausted from prolonged fasting.

This loss in weight, as Dr. Allen noted in his most severely sick animals, is another one of the important points to be considered. Dr. Allen points out that loss in weight occurring during fasting should not be corrected; the patient is to be allowed to remain below his previous weight, even becoming fairly thin. The attempt to increase his weight and thus to still further increase his metabolic activity results usually in a return of the diabetic condition.

The associated weakness which many of the patients exhibit is another one of the questions still unsolved—Whether or not the patient, although remaining thin, can recover his full strength? In this regard the suggestion of Dr. Barker[2] is opportune, viz.: that the harmful effects of under-weight upon the nervous system are not to be entirely neglected.

Our 14 cases comprised 3 mild glycosurias, 8 moderately severe, and 3 severe diabetics. We were successful in rendering them all sugar-free with the exception of 2 of the most severe cases. It is possible that these 2 might have become sugar-free, but in our inadequate experience we were loath to prolong their fasting beyond the seventh day. One other case remained sugar-free only twenty-four hours and we were unable again to render him absolutely free from sugar. In this case, however, we had reason to believe that he obtained bread and apples stolen from the trays of his neighbors in the ward. The majority of our cases complained rather bitterly of hunger toward the end of their fasting, although stating that they were not uncomfortable during the first few days. Several of the moderately severe

[1] Discussion of paper before a meeting of Suffolk County Medical Society in connection with Boston Medical Library, Dec. 2, 1914.

[2] Discussion on Diabetes. Association of American Physicians; Society Proceedings, Jour. Amer. Med. Assoc., 1915, vol. lv, p. 51.

cases referred to a general feeling of well-being and increased strength and brightness during the fasting; this in contrast to their previous feelings of depression and weakness. Results such as these are in accord with the results of other observers, tending to show that the weakness and depression of the diabetic state is due to the toxemia which disappears under the prolonged fasting. The majority of the patients remained sugar-free for from two days to two weeks following a return to food, although several of the earlier cases showed some sugar for a day or so following the giving too suddenly of a large amount of bread and a too rapid increase in their carbohydrate allowance. A day or so of fasting or a vegetable day was in most cases sufficient again to render them sugar-free. All of our patients showed a reduction in the ammonia content and the acidosis, with the exception of the 2 severe cases already referred to. In fact, the constantly severe ketonuria (high ammonia figure, strong diacetic and acetone reactions) was the impelling argument for putting them back on food. As we have said, it is possible, had we persevered in their fasting, that their acidosis too would have diminished.[1] Most of the patients remained weak following their fast. In fact, most of them still complain of easy tire and fatigue, even on dismissal from the hospital. One woman patient, a cook, complained of weakness for three months and over after returning home.

As far as the after-care and subsequent conditions of these patients are concerned, unfortunately we are without much data, except in two instances. As was to be expected, in these County patients it was extremely difficult to follow them after discharge from the hospital. In the two instances where we were able to get information they are still sugar-free after an interval of four and seven months respectively, are back at their regular occupations, are only slightly underweight, and are in apparently good condition. Subjectively, they state that they feel better than they have in years.

[1] Joslin, Brigham, and Hornor (The Boston Medical and Surgical Journal, March 16, 1916), in their article, "An Analysis of Fourteen Cases of Diabetes Mellitus Unsuccessfully Treated by Fasting," discuss somewhat similar cases.

No. Name Mrs. K. Case C. C. H.

1056

Date	Vol	Sp. Gr.	Reac	Alb	Acetone	Diacetic Acid	Beta-oxybutyric Acid	Nitrogen	Ammonia: Total	Ammonia: % of Nitrogen	Fehling's	Fermentation	Rotation: Before Fermentation	Rotation: After Fermentation	Urine: Total Sugar	DIET: Carbohydrate	DIET: Protein	DIET: Nitrogen	DIET: Fat	DIET: Alcohol	DIET: Calories	Carbohydrate Balance	$NaHCO_3$	Weight of Patient
10-5-15	960	1025	acid	Trace	+	0			.41	.043					11.42	Full diet								193¼
10-6	1440	1030	"	Trace	+	0			.734	.05					21.69	Starvation								
10-7	1020	1020	"	0	0	0			.66						0	Starvation								
10-8	1140	1019	"	0	Trace	0			.496						0	76.2	32.3		23.56		654.8			
10-9	720	1022	"	0	+	0			.517						0	66.3	26.49		19.96		462.8			
10-10	600	1026	"	0	++	0			.73						0	76.4	76		33.42		795.18			
10-11	540	1028	"	0	+	0			.42						0	109	56.37		49.99		1111.39			
10-12	540	1024	"	0	+	0			.5						0	94.6	57.58		68.92		1239			188
10-13	540	1024	"	0	+	0			.61						0	86.15	68.12		85.04		1382.94			
10-14	1020	1021	"	0	Trace	0			.417						0	97.15	70.94		85.46		1441.5			187
10-15	720	1027	"		Trace	0			.34						0	106.95	74.16		87.53		1488.			
10-16	720	1024	"		Trace	0			.216						0									186

Fig. 209.—Case I (Mrs. K., C. C. H.). Diabetic chart of case of mild diabetes. Patient became sugar-free in twenty-four-hour complete fast. Remained sugar-free until discharge from hospital (two weeks) upon a diet containing 107.95 gm. carbohydrate.

The following 2 cases will illustrate some of the points we have referred to:

Case I (Mrs. K.).—The first chart is that of a woman forty-two years of age, Austrian by birth, housekeeper by occupation, came to the hospital on October 4, 1915. She stated that for two and one-half years she thought she had been losing her strength; she complained of extreme tire on slight exertion and noticed that her appetite was becoming larger. In addition, she complained of occasional headache and a feeling of weakness and nervousness. The above symptoms gradually became worse, until now it is impossible for the patient to work for any considerable length of time without frequent periods of rest. Her appetite has increased progressively. The headaches have become more frequent and now she has attacks either in the morning or evening of each day. She has not noticed any great increase in the amount or frequency of urination. She has lost somewhat in weight during the past two years, although she is still a rather fleshy individual.

Past history is negative; usual diseases of childhood; she had malaria four years ago in New Mexico.

Family History.—She has been married sixteen years; three healthy children, no miscarriages. There is no history of diabetes in any other member of the family.

Physical examination reveals a large, fleshy, middle-aged woman, sitting quietly in bed with no apparent discomfort. Pulse somewhat fast, but regular, full and easily compressible. Her face is rotund and florid.

Eyes small.

Neck is rather short and thick. Thyroid distinctly palpable. A small palpable round enlargement in both right and left thyroid lobes.

Chest negative; heart and lungs negative; abdomen likewise negative. Spleen and liver not palpable. Extremities negative.

Reflexes present and brisk throughout.

Pelvic examination negative.

Blood-pressure: Systolic, 130; diastolic, 95.

Blood-count: 4,600,000 reds; hemoglobin 90; 8500 whites.

On the second day of a general ward diet she passed 1440 c.c. of urine, with a specific gravity of 1030, containing 1.5 per cent. of sugar; total sugar, 21.69 gm.; slight trace of acetone; no diacetic acid; 0.734 gm. of ammonia in twenty-four-hour specimen.

Within twenty-four hours of complete fast she became entirely sugar-free, the total amount of urine decreasing to 1120 c.c., specific gravity of 1020, no diacetic acid, no acetone, ammonia 0.66 gm.

During the balance of her two weeks' stay in the hospital her diet was increased progressively according to the table, so that at the end of ten days she was receiving 74.16 gm. of protein, 107.95 gm. of carbohydrate, 87.53 gm. of fat; total calories, 1488.2.

The analysis corresponding to this diet on the 16th showed 720 c.c. urine, specific gravity 1024, no sugar, trace of acetone, no diacetic, ammonia 0.62.

During the four months that have intervened since her discharge her urine has been seen on an average of once or twice a month, and at no time has shown sugar.

Clinically, the patient is in better condition than she has been for a number of years; she is able to do her work without tire; headaches have disappeared; she has lost altogether about 15 pounds in weight, and, in general, is in excellent condition.

Case II (A. S.).—The second patient, a more severe case of diabetes, is a carpenter, thirty-three years of age, Norwegian, came into the hospital November 19th, with the following history: Patient stated that he had always been well up to two months ago when he first noticed difficulty in retaining his urine. His urinary output increased considerably and he was constantly thirsty. He passed large amounts of urine at frequent intervals, more in the daytime he thinks, although he had to arise five or six times during the night to urinate. Four weeks ago he noticed pain in his legs. These pains he describes as "shooting" in character, starting from the knee and passing to the ankle. In addition to cramps in the legs in the early morn-

ing hours he had a sense of weakness in his lower extremities. He also complained of severe frontal headaches in the morning. Otherwise his general condition has been good with the exception of a slight loss in weight in spite of an extremely good appetite.

Past history is negative, except that he has suffered from frequent "colds."

Venereal history negative.

Family History.—Patient is single. Parents, one brother, and sister living and well.

Habits.—He drinks from two to three glasses of beer and one glass of whisky daily; smokes a little and chews a good deal.

Physical Examination.—Patient is tall and slender, well developed, but rather a thin young man of thirty-three. His face is flushed. His pupils are equal and react to light and accommodation.

Mouth negative, except tonsils are moderately enlarged. Neck negative; thyroid not enlarged.

He has a soft-blowing systolic murmur at the apex of the heart.

Abdomen is negative. Liver and spleen not palpable.

Blood-pressure: Systolic, 120; diastolic, 100.

Reflexes normal.

The first twenty-four-hour collection of urine on a full ward diet (Nov. 21st) showed nearly 6 liters, 5800 c.c., specific gravity 1035, 5.5 per cent. of sugar, trace of acetone, no diacetic, 2.55 gm. of ammonia.

Complete starvation, with the exception of whisky and coffee every two hours, reduced the urine in forty-eight hours to 2100 c.c., 1017 specific gravity, 1.75 per cent. sugar, 1.9 gm. ammonia.

In another twenty-four hours (four days after initial fast) the urine was sugar-free; the ammonia 1.4 gm.

The patient remained in the hospital until February 2d; he ran an occasional trace of sugar from time to time following the surreptitious taking of bread off other patients' trays. Upon discharge the urine was sugar-free, but the patient's tolerance to carbohydrate remained between 40 and 50 gm.

The method which we have used in the treatment of these cases follows rather closely that suggested by Dr. Elliott P. Joslin, of Boston.[1] In substance his method is as follows:

After entrance into the hospital the patient is put on the regular ward diet for two days to standardize the severity of the case, during which time complete twenty-four-hour urines are collected in a sterile container under toluol; the specimens examined for quantitative sugar, acetone, diacetic acid, and ammonia. After this the patient is put to bed and starved until sugar-free. After becoming sugar-free he is placed on green vegetables and gradually his tolerance for carbohydrate, protein, and fat is determined. Any return of sugar is a signal for a repetition of a twenty-four-hour fast, and even in the absence of glycosuria, weekly fast-days are instituted.

The following actual outline of this method as used in hospital practice may be found useful. The advantage of this outline lies in the fact that by it one may automatically increase the patient's diet progressively without the necessity each day of figuring up the requisite number of grams of protein, fat, and carbohydrate for each individual patient. While this outline cannot be followed dogmatically in every case, its use, or the use of some similar guide, will be found very largely to obviate the daily time-consuming task of arranging each individual patient's dietary. In fact, with such an outline the patient's diet can be almost entirely turned over to the dietitian, so that, unless glycosuria occurs, or for any other reason the patient does not do well, at the end of two weeks the patient has been carried to an extremely liberal diet, viz., protein, 70 gm.; fat, 200 gm.; carbohydrate, 100 gm. As will be seen, the method consists of daily increases in protein, fat, and carbohydrate parallel to the number of days' freedom from sugar after the initial fast. In the event of a return of sugar, ketonuria, or other complication, or if the patient is not doing well clinically, any change in diet can be made by simply ordering the patient returned to Diet No. 1 or 2, or whichever one is desired, whereupon merely reference to

[1] "Present-day Treatment and Prognosis in Diabetes," Elliott P. Joslin, Amer. Jour. Med. Sci., Oct., 1915.

the table will show at a glance the content of food elements in that particular diet. This method of daily increase is similar to the various dietary plans (Leube, Lenhartz) used in the medical treatment of chronic ulcer of the stomach.

Modification of Allen Method for Diabetic Patients, Arranged by Miss Maude E. Perry, Dietitian to the Michael Reese Hospital.

Diet No. 1.—Upon entrance to the ward place patient on regular ward diet for two days; during this period collect daily complete twenty-four-hour specimens of urine under toluol, examine for quantitative sugar, acetone, diacetic acid, and ammonia.

Diet No. 2.—Third day put patient to bed; give *absolutely nothing by mouth* except 3 to 4 ounces of the following liquids every two hours, from 7.00 A. M. to 7.00 P. M.: Black coffee; clear tea; chicken, mutton or beef broth; water ad lib. In the event of marked acidosis or impending coma ½ to 1 ounce of brandy or whisky may be added every two to four hours. Continue above until patient becomes sugar-free (from three to ten days).

Diet No. 3.—When urine is twenty-four hours sugar-free, *add* 150 gm. of 5 per cent. vegetables, viz., lettuce, spinach, string beans, celery, asparagus, cucumbers, Brussels sprouts, sorrel, endive, dandelion greens, cauliflower, tomatoes, egg plant, beet greens, water cress, cabbage, radishes, etc. Protein, 8.2 grams; fat, 0 grams; carbohydrate, 5.0 grams; calories, 54.[1]

Diet No. 4.—Urine two days sugar-free, *add* 150 grams 5 per cent. vegetables and two eggs.

Protein, 22.5 grams; fat, 10 grams; carbohydrate, 10.1 grams; calories, 234.[1]

Diet No. 5.—Urine three days sugar-free, *add* one-half grapefruit and 55 grams of lean meat.

[1] Daily diets corresponding to the above outline may be found at the conclusion of this article.

Protein, 37.6 grams; fat, 15.5 grams; carbohydrate, 15.2 grams; calories, 364.[1]

Diet No. 6.—Urine four days sugar-free, *add* 150 grams 5 per cent. vegetables and 55 grams of lean meat.

Protein, 55.1 grams; fat, 21 grams; carbohydrate, 20.3 grams; calories, 504.[1]

Diet No. 7.—Urine five days sugar-free, *add* 75 grams of 10 per cent. vegetables (onions, squash, turnip, carrots, mushrooms, beets) and 25 grams of lean meat, and 50 grams of cottage cheese or equivalent protein food.

Protein, 70.7 grams; fat, 24.5 grams; carbohydrate, 27.3 grams; calories, 631.[1]

Diet No. 8.—Urine six days sugar-free, *add* 30 grams of butter.

Protein, 70.7 grams; fat, 49.5 grams; carbohydrate, 27.3 grams; calories, 836.3.[1]

Diet No. 9.—Urine seven days sugar-free, *add* 75 grams of 10 per cent. fruit (lemons, oranges, cranberries, strawberries, blackberries, gooseberries, peaches, pineapple, watermelon) and 2 ounces of 20 per cent. cream, 5 grams of butter, substitute 20 grams of bacon for 30 grams of lean meat.

Protein, 70.1 grams; fat, 72.5 grams; carbohydrate, 34.3 grams; calories, 1131.[1]

Diet No. 10.—Urine eight days sugar-free, *add* ½ ounce of olive oil and 10 grams of butter.

Protein, 70.2 grams; fat, 96 grams; carbohydrate, 34.3 grams; calories, 1393.[1]

Diet No. 11.—Urine nine days sugar-free, *add* 50 grams of 15 per cent. vegetables (green peas, artichokes, parsnips, canned lima beans), 2 ounces of cream, 10 grams of butter, one egg; diminish lean meat 35 grams.

Protein, 70.9; fat, 122 grams; carbohydrate, 38.2 grams; calories, 1550.[1]

Diet No. 12.—Urine ten days sugar-free, *add* 25 grams of bread, 10 grams of butter, 1 ounce of cream, 15 grams of 10

[1] Daily diets corresponding to the above outline may be found at the conclusion of this article.

per cent. nuts (Brazil nuts, black walnuts, hickory, pecans, filberts).

Protein, 78.2 grams; fat, 166 grams; carbohydrate, 60.8 grams; calories, 2112.[1]

Diet No. 13.—Urine eleven days sugar-free, *add* ½ ounce olive oil, 10 grams of butter, 25 grams of bread; *diminish* meat 20 grams or *discontinue* cheese (protein too high).[1]

Diet No. 14.—Urine twelve days sugar-free, *add* 10 grams of butter, 75 grams of potato, and 15 grams of bacon.[1]

Diet No. 15.—Urine thirteen days sugar-free, *add* 25 grams of bread.[1]

Diet No. 16.—Urine fourteen days sugar-free, *add* 25 grams of potato.[1]

Protein	70 grams
Fat	200 "
Carbohydrate	100 "
Total calories	2480 "

The above summary is the average diet for mild cases of diabetes in patients averaging 70 kilograms in weight.

The following modification is arranged for *severe cases;*

Proceed in the same manner as above with the exception of the carbohydrates, unless, of course, glycosuria occurs.

Urine ten days sugar-free, *add* 10 grams of bread.

Urine eleven days sugar-free, no change in carbohydrate.

Urine twelve days sugar-free, *add* 10 grams of bread.

Urine thirteen days sugar-free, no change.

Urine fourteen days sugar-free, *add* 25 grams of potato.

Proceed in this manner, making the change in diet every other day, alternating, adding 25 grams of potato or 10 grams of bread as shown, until patient is getting 100 to 150 grams of potato and 70 to 80 grams of bread, or until carbohydrate tolerance is reached.[1]

The above modification of Miss Perry's follows in general lines the convenient little forms arranged by Dr. Joslin and

[1] Daily diets corresponding to the above outline may be found at the conclusion of this article.

published by Thomas Groom & Company of Boston. I would like to quote the following from their card:

"*Reappearance of Sugar.*—The return of sugar demands fasting for twenty-four hours or until sugar-free. The diet is then increased twice as rapidly as before, but the carbohydrate should not exceed half the former tolerance until the urine has been sugar-free for two weeks, and it should not then be increased more than 5 grams per week.

"*Weekly Fast Days.*—Whenever the tolerance is less than 20 grams carbohydrate, fasting should be practised one day in seven; when the tolerance is between 20 and 50 grams carbohydrate, upon the weekly fast day 5 per cent. vegetables and one-half the usual quantity of protein and fat are allowed; when the tolerance is between 50 and 100 grams carbohydrate, the 10 and 15 per cent. vegetables are added as well. If the tolerance is more than 100 grams carbohydrate, upon weekly fast days the carbohydrate should be halved."

The patient should have one day of restricted diet each week, no matter how mild the case is, for the purpose of still further sparing the already diminished function which controls carbohydrate metabolism. To accomplish this the patient is instructed upon discharge from the hospital either to examine his own urine once or twice weekly or to send specimens to the laboratory for examination. The patient is cautioned to report regularly from time to time, and is impressed with the fact that some supervision will probably always be necessary. This supervision as the patient remains sugar-free can be lengthened from weeks to months. In addition to care as to diet the patient's general hygiene and method of life should be inquired into and regulated as far as possible. He should be cautioned to abstain from excessive fatigue, to work and play well within his own limits of tire, to avoid excesses in all directions. If possible, a rest period in the middle of the work-day is a most valuable aid to this end. While his weight need not come up to its former level, still it should not become progressively less, but he should remain at a fairly stationary level, although this may be considerably below what he has considered his normal weight. Complications, such as extreme pruritus, edema, neuritis, neuroretinitis, furuncles and carbuncles, etc., are treated along usually accepted lines. Alkalies may be given in small amounts as increasing acidosis or impending coma warrant.

It is too short a time to speak of ultimate results, but from the experience already had this much appears true: The patients remain sugar-free; remain stationary at a somewhat reduced weight; feel physically much improved and are able to carry on their regular occupations and go about their daily routine in a relatively much better condition than before.

In a recent letter (Feb. 7, 1916) from Dr. Allen, he states his experience up to date as follows:

"Our youngest patient is twenty-six months, the oldest, sixty-nine years. It still apparently remains true that any case of uncomplicated diabetes can be cleared up. The young cases with coma threatened, which are the ones most physicians would be most afraid to starve, are the ones who generally improve most during the fast. The condition of nausea and weakness mentioned in some of the papers should be watched for, perhaps most especially in old persons who have had their diabetes a longer time. There have been a few terribly severe cases seen by others than myself, who have shown the 3.65 ratio on fasting and have died.

"As to ultimate prognosis, our longest experience is not quite two years. So far it has been unqualifiedly favorable. The patients, with very few exceptions, have followed their diet whether they are rich or poor; they do not seem to be going downhill, and many of them have shown a very perceptible improvement during the time at home as well as in the hospital. Children have done as well as grown people of similar severity, but, of course, the ultimate question of their growth and health remains entirely unsettled."

In regard to our extremely severe cases and cases of long standing, I recall a patient, fifty-five years of age, who had an extremely severe diabetes of fifteen years' duration. During this time he had been seen by the foremost diabetes authorities in this country and abroad; had submitted himself to various treatments, and had never been rendered sugar-free. This patient was seen in consultation, and although the Allen treatment was recommended, it, unfortunately, was not carried out, so that the result in this most interesting case, so far as the Allen treatment is concerned, could not be ascertained. The patient himself was not willing to submit to radical starvation and was only willing to have his carbohydrates very gradually reduced under careful hospital observation, together with the giving of large doses of alkalies. Under this regimen he did not become sugar-free, but did develop a terrify-

ing generalized anasarca which later disappeared with the giving of more carbohydrate and the diminution of alkalies.

The results obtained in these severe cases of diabetes of long standing, will, of course, be the real test of the Allen treatment. Men of the widest experience have reported deaths in the terribly severe cases following fasting, and it is these cases for which probably a special method of treatment must be devised. Dr. Elliott P. Joslin, of Boston, in a recent letter (Nov. 18, 1915) advises proceeding with great caution in these severe cases, and has suggested the following method of handling them:[1]

"The treatment which I recommend is that you do not interfere at all with the man's present habits of living; that is, getting up, going to bed, time spent at meals, time spent outdoors, etc.

"Second: That you do your best to find out what his diet is, only taking the greatest possible pains not to allow him to change it in the slightest possible degree. Do not let him try to diet strictly just to show you he is an upright diabetic.

"Third: At the end of twenty-four hours decrease the quantity of fat in his diet 25 grams, and continue to decrease it by that amount daily until he is having no fat. During all this time allow him to do just what he is at present, only perhaps very, very slightly restricting the total amount of exercise.

"Fourth: If he is having no sodium bicarbonate, give him none. If he is having it, plan to decrease it at the rate of 10 per cent. a day until it is wholly omitted. Remember that 1 quart of French Vichy contains about the equivalent of 4 grams bicarbonate of soda.

"Fifth: Though you decrease the soda, do not decrease the salts in his diet. Be sure he gets sufficient either in the form of broths (which probably contain about 0.9 per cent. salt) or by using freely with his food; or, if you wish, you could let him have a mineral water which contains little alkali, but does contain mixed salts.

"Sixth: It is my impression that at the time the fat is omitted the acidosis will have in great part decreased. If he is taking considerable soda, however—for example, as much as 8 grams—by that time very likely there would be a strong reaction for diacetic acid.

"Seventh: So soon as the fat is out of the diet take three days to get rid of your protein and simultaneously decrease the carbohydrate 25 grams a day providing he is having quite a little, but not below 40 grams, until the protein is omitted, and then halve the carbohydrate daily until you get to 10 grams. Probably your patient will either then be sugar-free or will become so after one day's fast.

[1] At the time of writing, Dr. Joslin was carrying out this plan of treatment, but had not published it. Since then two articles of his have appeared in which this method of treatment is still further discussed, viz., March number of the Amer. Jour. Med. Sci., and March 16th number of the Boston Med. and Surg. Jour.

"You need a first-class nurse and one who has seen a lot of diabetic patients of a severe type. One must be on the alert in handling these severe cases; one should see the patient twice a day, taking care, however, not to alarm him. He must be sure that the patient will not break over his diet and consider himself well when he is sugar-free and free from acid. He must be sure that some one who lives with him carries out your ideas until he and his family are thoroughly trained in what to do."

There are several interesting practical problems with reference to prolonged fasting in diabetes in general, particularly the point as to whether it is really advisable to render these patients sugar-free. While it has been conclusively proved that diabetics, even of the severest type, *may be* rendered sugar-free by the Allen method, it will probably take several years to determine whether or not these patients do best in the long run under such conditions. Many chronic diabetics of advancing years, particularly those patients who show in addition to their diabetes sclerotic changes in their cardiovascular and renal tissues, continue in active life and in seemingly good health in spite of their having from 0.75 to 1.5 per cent. or more of sugar; and, although you can render such patients sugar-free and keep them sugar-free, still, if you do so, they are underweight, they complain of weakness and are easily fatigued, and state that they do not feel so well as when they are enjoying a more liberal diet though they pass a small quantity of sugar from time to time. I believe the real question of the advantages of any method which will render diabetics sugar-free must rest upon the outcome of such patients over a long period of years. While theoretically, of course, and experimentally, as Dr. Allen has shown, glycosuria should be cleared up and the sugar-free condition maintained in order that the diminished pancreatic function may be protected as far as possible, still only by this experience and observation of many patients over a considerable period of time can the ultimate decision be reached.

I have in mind particularly a physician, fifty years of age, who has had glycosuria during the past nine months. It is extremely easy to render him sugar-free, but when sugar-free he loses weight, he looks badly, he cannot stand the fatigue of the operating room, complains of extreme tire and exhaustion, and

his friends and family say he looks "gray and poorly." Allow him to eat a moderate amount of bread and a more generous diet in general and he looks better, he feels better, and is able to get through his work without discomfort. To me it is an open question whether one should insist upon rigid adherence to a diet which will keep the urine sugar-free, or to allow him to eat those things on which he feels the best himself. In fact, the decision is often made by the patient himself, who, notwithstanding advice to the contrary, asserts that he intends to "forget" his diabetes and will eat what he pleases.

SPECIMEN DIABETIC DIETS—ALLEN METHOD[1]

Moderate or Mild Cases

Arranged by MISS MAUDE E. PERRY, Dietitian to the Michael Reese Hospital.

Patient, 70 kilograms in weight.

Diet No. 3.—Urine twenty-four hours sugar-free: Protein, 8.2 gm.; fat, 0 gm.; carbohydrate, 5.0 gm.; calories, 54 (approx.).

	Protein (gm.).	Fat (gm.).	Carbohydrate (gm.).	Calories
Breakfast:				
One cup of black coffee.				
10 A. M.:				
4 ounces of clear broth	2.8	..	..	12
Dinner:				
Asparagus tips, 50 gm.	0.8	..	1.7	10
Lettuce, 25 gm.	0.4	..	0.8	5
Coffee.				
3 P. M.:				
Clear broth, 4 oz.	2.8	..	..	12
Supper:				
String beans, 50 gm.	1.0	..	1.7	10
Celery, 25 gm.	0.4	..	0.8	5
Coffee.				
Total	8.2	..	5.0	54

[1] These diabetic diets, corresponding to the daily progressive diets, are merely offered as *specimen* diets, not necessarily to be followed exactly, but which can be varied to meet the requirements of individual diet kitchens. For instance, any of the 5 per cent. vegetables can be substituted for those listed above.

Diet No. 4.—Urine two days sugar-free: Protein, 22.5 gm.; fat, 10.0 gm.; carbohydrate, 10.1 gm.; calories, 234.

	Protein (gm.).	Fat (gm.).	Carbohydrate (gm.).	Calories.
Breakfast:				
One egg	6.0	5	..	75
Rhubarb, 50 gm.	0.8	..	1.7	10
Coffee.				
10 A. M.:				
Broth, 4 oz.	2.8	..	...	12
Dinner:				
Cauliflower, 50 gm.	0.8	..	1.7	10
Tomatoes, 75 gm.	1.3	..	2.5	15
Lettuce, 25 gm.	0.4	..	0.8	5
Coffee.				
3 P. M.:				
Broth, 4 oz.	2.8	..	...	12
Supper:				
One egg	6.0	5	...	75
Asparagus tips, 50 gm.	0.8	..	1.7	10
Spinach, 50 gm.	0.8	..	1.7	10
Coffee.				
Total	22.5	10	10.1	234

Diet No. 5.—Urine three days sugar-free: Protein, 37.6 gm.; fat, 15.5 gm.; carbohydrate, 15.2 gm.; calories, 364.

	Protein (gm.).	Fat (gm.).	Carbohydrate (gm.).	Calories.
Breakfast:				
Grapefruit, one-half	...	...	5.0	20
One egg	6.0	5.0	...	75
Coffee.				
10 A. M.:				
Broth, 4 oz.	2.8	...	...	12
Dinner:				
Sauerkraut, 50 gm.	0.8	...	1.7	10
Green beans, 50 gm.	1.0	...	1.7	10
Tomato salad, 50 gm.	0.8	...	1.7	10
Roast beef, 55 gm.	15.0	5.5	...	110
Coffee.				
3 P. M.				
Broth, 4 oz.	2.8	...	...	12
Supper:				
One egg	0.6	5.0	...	75
Cauliflower, 50 gm.	0.8	...	1.7	10
Lettuce, 50 gm.	0.8	...	1.7	10
Rhubarb, 50 gm.	0.8	...	1.7	10
Coffee.				
Total	37.6	15.5	15.2	364

Diet No. 6.—Urine four days sugar-free: Protein, 55.1 gm.; fat, 21 gm.; carbohydrate, 20.3 gm.; calories, 504.

Breakfast:	Protein (gm.).	Fat (gm.).	Carbohydrate (gm.).	Calories.
Grapefruit, one-half	...	...	5.0	20
One egg	6.0	5	...	75
Lean ham, 30 gm.	8.0	3	...	60
Coffee.				
10 A. M.: Broth, 4 oz.	2.8	..	...	12
Dinner: Roast lamb, 50 gm.	14.0	5	...	100
Brussels sprouts, 100 gm.	1.7	..	3.4	20
Celery, 50 gm.	0.8	..	1.7	10
Rhubarb, 50 gm.	0.8	..	1.7	10
Water-cress, 50 gm.	0.8	..	1.7	10
Coffee.				
3 P. M.: Broth, 4 oz.	2.8	..	...	12
Supper: Cold chicken, 30 gm.	8.0	3	...	60
Egg-plant, 50 gm.	0.8	..	1.7	10
Spinach, 100 gm.	1.8	..	3.4	20
Tomato salad, 50 gm.	0.8	..	1.7	10
One egg	6.0	5	...	75
Coffee.				
Total	55.1	21	20.3	504

Diet No. 7.—Urine five days sugar-free: Protein, 70.7 gm.; fat, 24.5 gm.; carbohydrate, 27.3 gm.; calories, 631.

Breakfast:	Protein (gm.).	Fat (gm.).	Carbohydrate (gm.).	Calories.
Grape-fruit, one-half	...	...	5.0	20
One egg	6.0	5.0	...	75
Fish, 30 gm.	8.0	3.0	...	60
Coffee.				
Dinner:				
Broth, 3 oz.	2.1	...	...	9
Steak, 75 gm.	20.0	7.5	...	150
Cabbage, 100 gm.	3.7	...	3.4	20
Green beans, 100 gm.	2.0	...	3.4	22
Lettuce salad, 50 gm.	0.8	...	1.7	10
Rhubarb, 50 gm.	0.8	...	1.7	10
Coffee.				
Supper:				
Lean cold meat, 35 gm.	9.0	3.5	...	70
Broiled onions, 75 gm.	1.8	...	5.0	28
Asparagus tips, 100 gm.	1.7	...	3.4	20
Escarole salad, 50 gm.	0.8	...	1.7	10
Cottage cheese, 50 gm.	10.0	0.5	2.0	52
One egg	6.0	5.0	...	75
Coffee.				
Total	70.7	24.5	27.3	631

Diet No. 8.—Urine six days sugar-free: Protein, 70.7 gm.; fat, 49.5 gm.; carbohydrate, 27.3 gm.; calories, 836.3

Same amounts of:
5 per cent. vegetables,
10 per cent. vegetables,
5 per cent. fruits,
meat,
eggs,
cheese,

plus 30 gm. butter.	Protein (gm.).	Fat (gm.).	Carbohydrate (gm.).	Calories.
Total	70.7	49.5	27.3	836.3

Diet No. 9.—Urine seven days sugar-free: Protein, 70.1 gm.; fat, 72.5 gm.; carbohydrate, 34.3 gm.; calories, 1131.

	Protein (gm.).	Fat (gm.).	Carbohydrate (gm.).	Calories.
Breakfast:				
Grape-fruit, one-half	...	...	5.0	20
Bacon, 20 gm	3.5	10.0	...	104
One egg,	6.0	5.0	...	75
Butter, 10 gm	...	8.0	...	72
Cream, ½ oz	0.5	3.0	0.5	30
Coffee.				
Dinner:				
Broth, 3 oz	2.1	...	...	9
Roast chicken, 75 gm	20.0	7.5	...	150
Squash, 75 gm	1.8	...	5.0	28
Tomatoes, 100 gm	1.7	...	3.4	20
Spinach, 50 gm	0.8	...	1.7	10
Lettuce, 50 gm	0.8	...	1.7	10
Gooseberries, 75 gm	1.8	...	5.0	28
Butter, 15 gm	...	13.0	...	120
Cream, ½ oz	0.5	3.0	0.5	76
Coffee.				
Supper:				
Cold beef, 30 gm	9.4	3.5	...	70
One egg	6.0	5.0	...	75
Cottage cheese, 50 gm	10.0	0.5	2.0	52
Green beans, 100 gm	1.7	...	3.4	20
Creamed celery, 50 gm	0.8	...	1.7	10
Rhubarb, 100 gm	1.7	...	3.4	20
Cream, 1 oz. (on celery)	1.0	6.0	1.0	60
Butter, 10 gm	...	8.0	...	72
Coffee.				
Total	70.1	72.5	34.3	1131

Diet No. 10.—Urine eight days sugar-free: Protein, 70.2 gm.; fat, 96 gm.; carbohydrate, 34.3 gm.; calories, 1393.

Same amount of:
5 and 10 per cent. vegetables and fruits,
Meat and cheese,
Eggs,
Cream,
add
Olive oil, ½ oz.,
Butter, 10 gm.

	Protein (gm.).	Fat (gm.).	Carbohydrate (gm.).	Calories.
Total	70.2	96	34.3	1393

Diet No. 11.—Urine nine days sugar-free: Protein, 70.9 gm.; fat, 122 gm.; carbohydrate, 38.2 gm.; calories, 1550.

	Protein (gm.).	Fat (gm.).	Carbohydrate (gm.).	Calories.
Breakfast:				
Same as two previous plus 5 gm. butter	10.0	30.5	5.5	342
Dinner:				
Broth, 3 oz.	2.1	..	...	9
Roast beef, 50 gm.	14.0	5	...	100
Green peas, 50 gm.	3.0	..	5.0	32
Cauliflower, 100 gm.	1.7	..	3.4	20
Egg plant, 100 gm.	1.7	..	2.0	20
Tomato salad, 50 gm.	0.8	..	1.7	10
Gooseberries, 75 gm.	1.8	..	5.0	28
Butter, 20 gm.	...	17	...	153
Olive oil, ½ oz.	...	15	...	135
Cream, ½ oz.	0.5	3	0.5	30
Coffee.				
Supper:				
Sardines, 25 gm.	6.0	6.0	...	78
One egg	6.0	5.0	...	75
Cottage cheese, 50 gm.	10.0	0.5	2.0	52
Mushrooms, 75 gm.	1.8	..	5.0	28
Asparagus tips, 100 gm.	1.7	..	3.4	20
Escarole salad, 50 gm.	0.8	..	1.7	10
Custard (1 egg, 2 oz. cream)	8.0	17	2.0	195
Butter, 20 gm.	...	17	...	153
Cream, 1 oz.	1.0	6	1.0	60
Coffee.				
Total	70.9	122	38.2	1550

Diet No. 12.—Urine ten days sugar-free: Protein, 78.2 gm.; fat, 166 gm.; carbohydrate, 60.8 gm.; calories, 2112.

	Protein (gm.).	Fat (gm.).	Carbohydrate (gm.).	Calories.
Breakfast:				
Same as day before, plus				
10 gm. bread,				
15 gm. butter,				
2 oz. cream	12.5	55.5	13.5	603.5
Dinner:				
Cream soup (2 oz. cream)	2.0	12	2.0	120
Roast lamb, 50 gm.	14.0	5	...	100
Lima beans, 50 gm.	3.0	..	5.0	32
Spinach, 100 gm.	1.7	..	3.4	20
Sauerkraut, 100 gm.	1.7	..	3.4	20
Water-cress salad, 50 gm.	0.8	..	1.7	10
Olive oil, ½ oz.	...	15	...	135
Gooseberries, 75 gm.	1.8	..	5.0	28
Butter, 25 gm.	...	21	...	189
Filberts, 15 gm.	3.0	10	1.0	108
Bread, 15 gm.	1.5	..	9.0	75
Cream, ½ oz.	0.5	3	0.5	30
Coffee.				
Dinner total	30.0	66	31	859
Supper:				
Cold chicken, 20 gm.	6.0	0.2	...	40
Cottage cheese ,50 gm.	10.0	0.5	2.0	52
One egg	6.0	5.0	...	75
Broiled onions, 75 gm.	1.8	...	5.0	28
Tomatoes, 100 gm.	1.7	...	3.4	20
String beans, 100 gm.	1.7	...	3.4	20
Custard	8.0	17.0	2.0	195
Butter, 20 gm.	...	17.0	...	189
Cream, ½ oz.	0.5	3.0	0.5	30
Coffee.				
Supper total	35.7	44.5	16.3	649
Daily total	78.2	166.0	60.8	2111.5

Diet No. 13.—Urine eleven days sugar-free:

Diet: Same amounts of vegetables, fruits, cream. Either diminish meat 20 gm. or discontinue cheese. Add ½ oz. olive oil, 10 gm. butter, 25 gm. bread.

Diet No. 14.—Urine twelve days sugar-free:

Diet: Same as day before plus 10 gm. butter, 75 gm. potato, 15 gm. bacon.

Diet No. 15.—Urine thirteen days sugar-free:
Diet: Add 25 gm. bread.
Diet No. 16.—Urine fourteen days sugar-free:
Diet: Add 25 gm. potato.

End of two weeks: Protein, 70 gm.; fat, 200 gm.; carbohydrate, 100 gm. (approx.).

Severe Cases

Proceed in the same manner as above with exception of carbohydrates, unless sugar develops.

Urine ten days sugar-free, add 10 gm. bread.
Urine eleven days sugar-free, no change in carbohydrate.
Urine twelve days sugar-free, add 10 gm. bread.
Urine thirteen days sugar-free, no change.
Urine fourteen days sugar-free, add 25 gm. potato.

Proceed in this manner, making the change in diet every other day; alternating, adding 25 gm. potato or 10 gm. bread, as shown, until patient is getting 100 to 150 gm. potato and 70 to 80 gm. bread, or until carbohydrate tolerance is reached.

SYNOPSIS OF PRECEDING TABLES

Adaptation of "Allen Method" for Diabetic Patients

MILD CASES. 70 KILOGRAMS IN WEIGHT.

Diet No. 1.—Entrance to hospital: Full diet. Examine urine for sugar, acteone, diacetic acid, ammonia.

Diet No. 2.—Starvation or fasting until sugar-free:
Water, tea, coffee, and clear meat broth, as desired.

Diet No. 3.—Urine twenty-four hours sugar-free:
Add 150 gm. of 5 per cent. vegetables.
Protein, 8.2 gm.; fat, 0 gm.; carbohydrate, 5 gm.; calories, 54.

Diet No. 4.—Urine two days sugar-free:
Add 150 gm. 5 per cent. vegetables and two eggs.
Protein, 22.5 gm.; fat, 0 gm.; carbohydrate, 10.1 gm.; calories, 234.

Diet No. 5.—Urine three days sugar-free:
Add one-half grape-fruit and 55 gm. of lean meat.
Protein, 37.6 gm.; fat, 15.5 gm.; carbohydrate, 15.2 gm.; calories, 364.

Diet No. 6 .—Urine four days sugar-free:
Add 150 gm. of 5 per cent. vegetables and 55 gm. of lean meat.
Protein, 55.1 gm.; fat, 21 gm.; carbohydrate, 20.3 gm.; calories, 504.

Diet No. 7.—Urine five days sugar-free:
Add 75 gm. of 10 per cent. vegetables and 25 gm. of lean meat (50 gm. of cottage cheese or equivalent protein food).
Protein, 70.7 gm.; fat, 24.5 gm.; carbohydrate, 27.3 gm.; calories, 631.

Diet No. 8.—Urine six days sugar-free:
Add 30 gm. of butter.
Protein, 70.7 gm.; fat, 49.5 gm.; carbohydrate, 27.3 gm.; calories, 836.3.

Diet No. 9.—Urine seven days sugar-free:

Add 75 gm. of 10 per cent. fruit and 2 oz. of 20 per cent. cream, 5 gm. butter.
Substitute 20 gm. of bacon for 30 gm. of lean meat.

Protein, 70.1 gm.; fat, 72.5 gm.; carbohydrate, 34.3 gm.; calories, 1131.

Diet No. 10.—Urine eight days sugar-free:

Add ½ oz. of olive oil and 10 gm. of butter.

Protein, 70.2 gm.; fat, 96 gm.; carbohydrate, 34.3 gm.; calories, 1393.

Diet No. 11.—Urine nine days sugar-free:

Add 50 gm. of 15 per cent. vegetables and 2 oz. cream, 10 gm. butter, 1 egg.
Diminish lean meat 35 gm.

Protein, 70.9 gm.; fat, 122 gm.; carbohydrate, 38.2 gm.; calories, 1550.

Diet No. 12.—Urine ten days sugar-free:

Add 25 gm. of bread and 10 gm. of butter, 1 oz. cream, 15 gm. of filberts.

Protein, 78.2 gm.; fat, 166 gm.; carbohydrate, 60.8 gm.; calories, 2112.

Diet No. 13.—Urine eleven days sugar-free:

Add ½ oz. olive oil, 10 gm. butter, 25 gm. bread. Diminish meat 20 gm. or discontinue cheese. . (Protein too high.)

Diet No. 14.—Urine twelve days sugar-free:

Add 10 gm. butter, 75 gm. potato.
Add 15 gm. of bacon.

Diet No. 15.—Urine thirteen days sugar-free:

Add 25 gm. bread.

Diet No. 16.—Urine fourteen days sugar-free:

Add 25 gm. potato.

Protein, 70 gm.; fat, 200 gm.; carbohydrate, 100 gm.; calories, 2480.

CLINIC OF DR. RICHARD J. TIVNEN

MERCY HOSPITAL

THE RELATION OF THE UPPER RESPIRATORY TRACT TO METASTATIC INFECTIONS

The Rôle Played by the Tonsils and the Nasal Accessory Sinuses as Atria of Infections. General Symptomatology, Etc.

March 1.

I THOUGHT it would be of interest to take up with you this afternoon a consideration of the upper respiratory tract in its relation to metastatic infections. The structures which we shall comprehend in the term "upper respiratory tract" are the nose, the nasal accessory sinuses, the throat, the tonsils, the mouth, and the teeth. Time will not permit an extensive discussion of all of these structures, nor, indeed, a detailed consideration of any particular one. We will devote ourselves, therefore, only to a survey of a number of them and refer to a number of illustrative cases which have come under our own observation and treatment, apropos to the structures under consideration, and refer in a practical way to such points of interest concerning these structures and these cases as our own experience has found of value and which will emphasize the general subject under consideration.

THE TONSIL

The tonsils as an atrium of infection in general systemic infections are so well recognized and accepted that only a brief reference is necessary. Jonathan Wright's aphorism that "the tonsillar crypts are test-tubes in which bacterial cultures are constantly opposed by the resisting power of the tissues" is, in the light of modern investigation of metastatic infections, a generally

accepted truism. Rosenow's work, in separating the different strains of infective organisms found in tissue and connecting these organisms with specific processes, notably the metastatic joint lesions, has established beyond question the long-suspected relation of tonsillar infection to metastatic joint infection, and placed this pathologic problem upon a certain and scientifically correct basis. This connection, clinically, had been noted from almost time immemorial, and it was an epoch-making scientific accomplishment to have these clinical observations corroborated by accepted scientific procedures.

As to the explanation of the tonsil being so often the cause of metastatic infection, no comprehensive data are at hand. The following factors, however, are of significance:

(1) The structure of the tonsil itself, a lymphoid structure in which numerous channels, lined by epithelium, ramify, pass completely through its substance, and open on its free surface—an arrangement that invites the collection of infective microorganisms and favors their retention and propagation.

(2) Their close relation to surrounding and neighboring tissues and structures, and intimate connection thereto by contact, continuity, lymphatic and vascular channels, etc.

(3) Their exposed position and very frequently diseased condition.

By common consent the tonsil is regarded as a very frequent, if not, indeed, the most frequent focal center for metastatic infection. The arthritides, in particular, in a large number of cases, admittedly, have their origin in tonsillar infection. From an operative standpoint, I have personally observed a large number of cases in which attacks of arthritis have disappeared after a complete enucleation of the tonsil, and from my own personal observation and experience I am thoroughly convinced that diseased tonsils are the most common source of arthritic infections. These particular cases are so frequent, and the metastases so well established, that we will pass this phase of the subject and direct your attention instead to a number of other metastatic manifestations of tonsillar disease which have come under our notice and which have not been so frequently referred to in the litera-

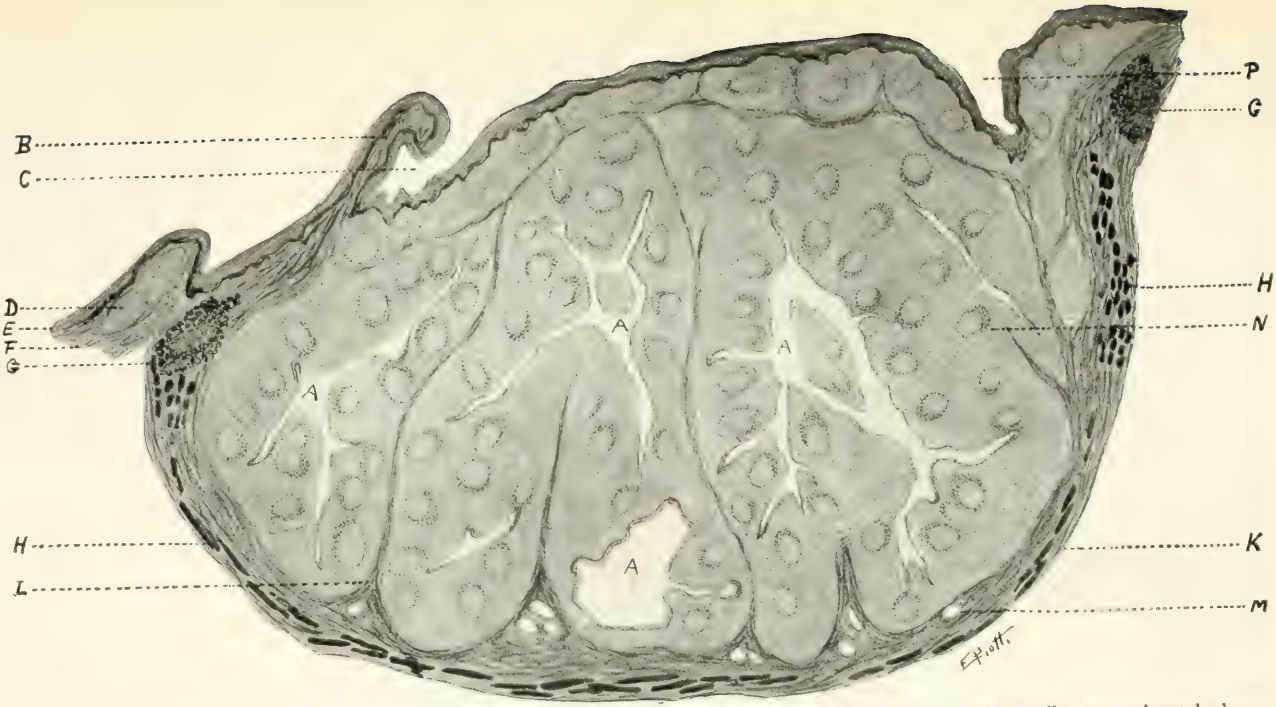

Fig. 210.—Cross-section of human tonsil, age eleven years, dissected out in capsule. (× 10.) This tonsil was moderately hypertrophied, with crypts greatly dilated and filled with detritus. It represents a condition found in a large percentage of the hypertrophied tonsils of childhood: *A*, Crypts, very irregular and dilated; *B*, plica triangularis; *C*, anterior fossa; *D*, lymphoid follicles outside capsule; *E*, surface epithelium; *F*, tunica propria of surface mucosa; *G*, mucous glands; *H*, muscle-fibers of capsule; *K*, capsule; *L*, trabecula; *M*, blood-vessel; *N*, lymphoid follicle with germinal center; *P*, posterior fossa. (From Barnes, "The Tonsils.")

ture. Before proceeding, however, I would like to refer briefly to the tonsil itself and its infections.

The tonsil, it may be emphasized, does not always give frank expression, either in its appearance or clinical history, that

Fig. 211.—The atrophic tonsil (twenty years). Lymphoid tissue reduced to a mere strip around the crypt. The remainder of the tonsil is a solid mass of fibrous tissue. In the lower quadrant are several nodules of bone and cartilage. (From Barnes, "The Tonsils.")

it is the seat of an infective process. The patient, for example, may give no history of sore throat and the tonsil itself may present to an inspection alone practically a normal appearance.

The consideration of the question whether a given tonsil is the source of the metastasis, or if, indeed, the tonsil itself is the seat of an infective process often requires some considerable investigation. Inspection alone in a large number of cases is not sufficient to establish the infective relationship. I have found it of advantage in my examination of such cases to consider three particular factors:

(1) The tonsillar history;

(2) Inspection of the tonsils;

(3) Expression of the crypts and bacteriologic examination of their contents.

I. *As to the History.*—This will often disclose previous attacks of tonsillitis, or present other suggestive facts, such as complaints of "irritation," "fulness," "discomfort," etc., in or about the tonsillar region—symptoms which indicate a subacute or chronic low grade infection of the tonsillar tissue.

II. *The Appearance on Inspection.*—The appearance of the tonsils and the tissues adjacent, as the palate and pharynx, are often only suggestive, but it is well to regard a well-defined congested appearance of the tonsillar pillars and surrounding tissues, distinctly demarkated, as strongly suspicious of such infections, even in the absence of a history of sore throat. The *size* of the tonsil itself is of importance. In my own experience I have come to regard the small compact fibrous gland, the so-called "submerged" or "buried" tonsil as a more frequent cause of metastatic infection than the large hypertrophied tonsil. I have reached the conclusion that at least one factor explaining this is that the size of the tonsil itself is an important aid in the emptying of the tonsillar crypts; a large tonsil, for example, in the act of deglutition presents a mass or bulk which is easily squeezed or "milked" by the muscles concerned in this act, and hence, in consequence, the infective material in the tonsillar crypts of such a tonsil is not permitted to be retained, propagated, or disseminated in the general system.

Apart from the fact that the faucial openings of the crypts of the so-called "submerged" tonsils are correspondingly small in caliber and more or less sealed or constricted, due to changes

which have resulted from previous inflammatory attacks, neither the tonsil nor the crypts of these small so-called "submerged" tonsils present a sufficient mass or bulk to be grasped or "milked" by the muscles of the pharynx, etc., during the act of deglutition, and, therefore, the crypts of such tonsils cannot be successfully emptied by this mechanism and retention, propagation and dissemination of pathogenic material in and from such crypts easily takes place.

III. *Expression of the Crypts and Bacteriologic Examination of Their Contents.*—Before concluding the examination of the tonsils the crypts should be expressed or "milked" and the expressed material subjected to bacteriologic examination. Such "expression" may disclose the fact that an unsuspected crypt harbors in its depths a large amount of infective material which a casual inspection of its surface alone did not disclose.

Apart from the arthritides it is well recognized also that tonsillar infections may be the cause of cardiovascular degeneration, Bright's disease, and many other metastatic processes. I have had a considerable number of cases of nephritis which cleared up in a most remarkable way after the tonsils had been removed.

A systematic plan of studying these more or less obscure cases from their etiologic aspect would indicate I am certain a more frequent causal relation between infections of the tonsils and cardiovascular degeneration and nephritic processes than is usually suspected. Within the last year I have had a number of cases of goiter which, in their clinical manifestation, seemed to indicate a very close relationship between the enlargement of the thyroid gland and infections of the tonsils. From among a number a brief reference to 2 cases which recently came under my observation will illustrate this connection. One, a patient referred to me by Dr. E. Wyllys Andrews, a woman of twenty-eight, married, presented a considerable enlargement of the thyroid gland, involving both lobes, from which she had suffered since the age of eighteen. Other than the cosmetic effect of this enlargement the patient had not experienced any difficulty whatever until recently. For a period approximating two months

previous to her visit she had begun to suffer with attacks of "smothering," and was unable to rest comfortably in bed on account of being troubled with a feeling of "suffocation." The goiter also had increased in size within this period. There was no history of sore throat, either within this period or previously. Inspection of the tonsils and expression of the crypts likewise failed to elicit a demonstrable pathologic finding. I enucleated her tonsils and following the removal there was instant and complete relief from the feeling of "suffocation" and "smothering." In addition, the size of the goiter noticeably decreased and became softer. This relief has been maintained for a period of some three months.

The second case was that of a patient of Dr. Joseph Miller. A woman, thirty-five years old, married, had follicular tonsillitis, involving both tonsils, of considerable severity, the bacteriologic finding being the staphylococci. Some weeks after the tonsillitis the patient began to suffer with nervousness, loss of weight and appetite, could not sleep and became irritable, and, generally, presented a marked "run-down" condition. Dr. Miller discovered an enlargement of the thyroid gland, centrally located. The patient stated that this enlargement of the thyroid developed since her attack of tonsillitis. After the removal of the tonsils the goiter has almost completely disappeared, the patient's condition has improved steadily, and at the present time—a period since the tonsillitis approximating four months—she is practically well.

NASAL ACCESSORY SINUSES

Let us next consider the rôle of the nasal accessory sinuses as factors in metastatic infections. That infection can be and is transmitted from the nasal accessory sinuses to neighboring and distant structures is already proved, both by clinical experiments and by analogy. The literature shows from year to year an increasing number of reported cases. Moreover, we are all familiar with the frequent association of systemic infection chargeable to oral infection, pyorrhea, alveolar and diseased teeth, tonsils, middle ear and mastoid infection, and by analogy

alone it is reasonable to assume that the nasal accessory sinuses

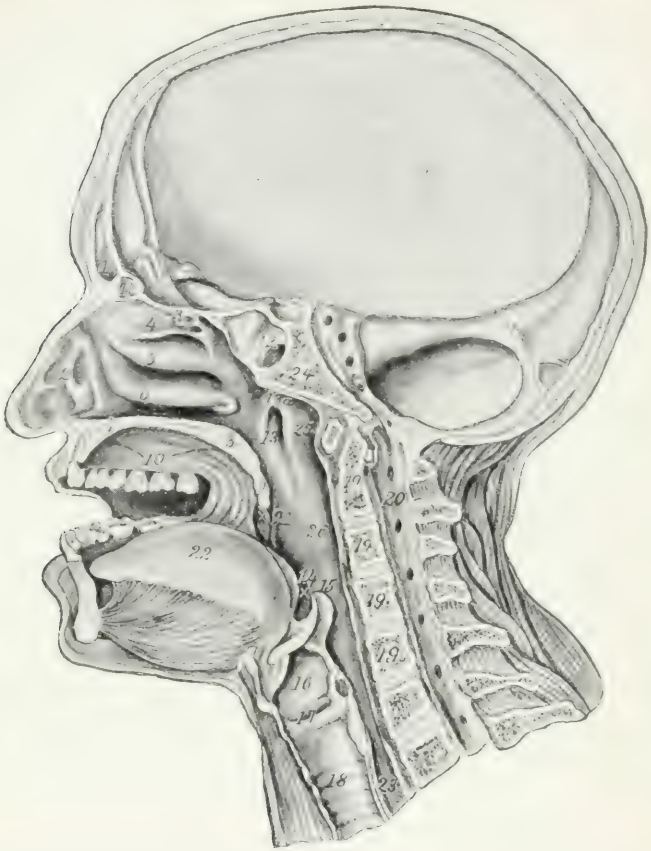

Fig. 212.—Sagittal section of head and neck: *1*, Frontal sinus; *2*, lateral cartilages of nose; *3*, fourth turbinate (not constant); *4*, superior turbinate; *5*, middle turbinate; *6*, inferior turbinate; *7*, hard palate; *8*, soft palate; *9*, uvula; *10*, arch of roof of mouth; *11*, anterior ethmoidal cells; *12*, sphenoidal sinus; *13*, Eustachian orifice; *13a*, position of tubal tonsil; *14*, position of lingual tonsil; *15*, epiglottis; *16*, vestibule of larynx; *17*, position of vocal cords; *18*, trachea, showing rings; *19*, bodies of cervical vertebræ; *20*, spinal canal, showing foramina of exit of nerves; *21*, position of faucial tonsil; *22*, tongue; *23*, esophagus; *24*, basilar process of occipital bone; *25*, nasopharynx; *26*, oropharynx. (From Kyle, "Diseases of the Nose and Throat.")

may likewise be the focal point for similar infective processes. At the Mercy Hospital, Chicago, in the service of Dr. Murphy, a

large number of joint infections are encountered. The amount of material is tremendous. Based upon a considerable number of observations of such cases, I have reached the conclusion that the

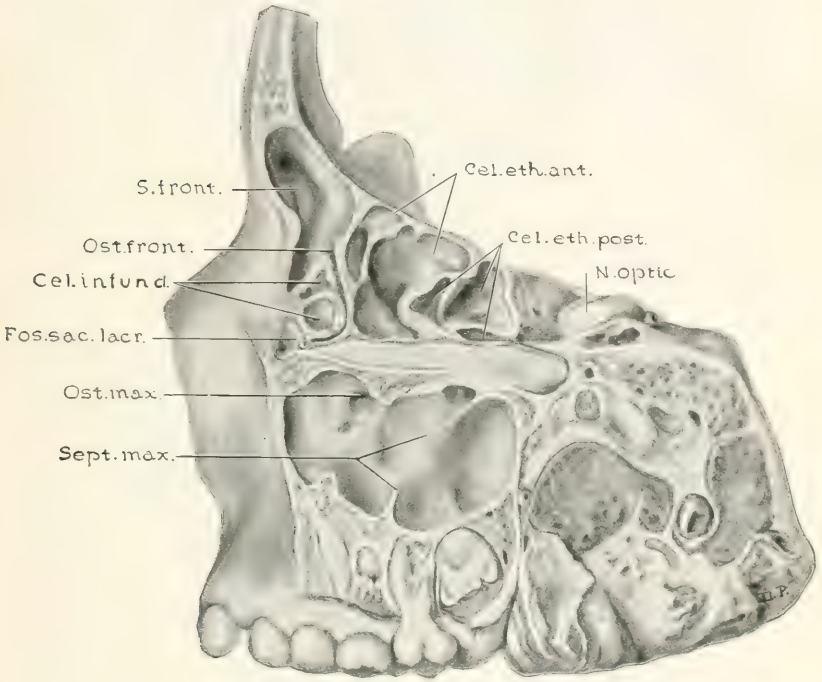

Fig. 213.—Specimen from a child eight years, eight months, and one day old. (Series D, No. 63.) Lateral view of frontal, ethmoidal, and maxillary sinus areas, the lateral portion of each having been removed by sagittal cuts. Note that the sinus frontalis developed directly from the infundibulum ethmoidale. Note also the incomplete septa in the sinus maxillaris: *Cel.eth.ant.*, Cellulæ ethmoidales anterior; *Cel.eth.post.*, cellulæ ethmoidales posterior; *N.optic.*, nervus opticus; *Sept.max.*, septulæ maxillares; *Ost.max.*, ostium maxillare; *Fos.sac.lacr.*, fossa sacci lacrimalis; *Cel.infund.*, cellulæ infundibulares; *Ost.front.*, ostium frontale; *S.front.*, sinus frontalis. (From Davis, "Development and Anatomy of the Nasal Accessory Sinuses in Man.")

nasal accessory sinuses as a focal center for general metastatic infections have not been given the prominence which they merit, and I am equally convinced that the future will establish the fact that the nasal accessory sinuses are an exceedingly common cause

for general metastatic processes, pressing indeed, in my judgment, the tonsils, very closely for the first position as agents in such pathologic processes.

When we consider the central location of these cavities, their number, their general arrangement and relatively large extent, their favorable situation for infection, their easily disturbed

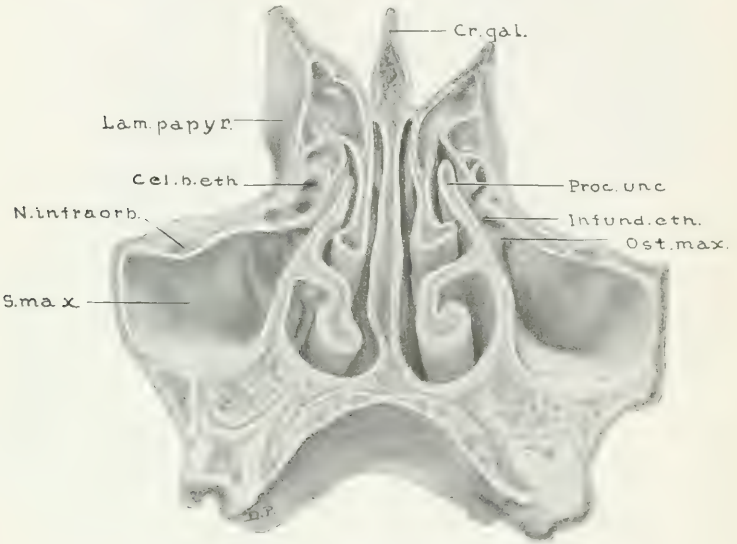

Fig. 214.—Specimen from a child nine years, ten months, and nineteen days old. (Series D, No. 65.) Anterior view of coronal section, cut 20 mm. posterior to the nasion, showing the size and relations of the sinus maxillaris, the ostium maxillare, and its manner of communication with the infundibulum ethmoidale, the bulla ethmoidalis, processus uncinatus; and cellulæ ethmoidales anterior: *Cr.gal.*, Crista galli; *Proc.unc.*, processus uncinatus; *Infund.eth.*, infundibulum ethmoidale; *Ost.max.*, ostium maxillare; *S.max.*, sinus maxillaris; *N.infraorb.*, nervus infraorbitalis; *Cel.b.eth.*, cellula bulla ethmoidalis; *Lam.papyr.*, lamina papyracea. (From Davis, "Development and Anatomy of the Nasal Accessory Sinuses in Man.")

drainage outlets, their consequent susceptibility to infection, their intimate connection with neighboring structures and the body generally, either by direct contact, continuity and contiguity, or vascular and lymphatic channels, we can better appreciate the likelihood of such infections, occurring individually or collectively, and can likewise more easily comprehend the

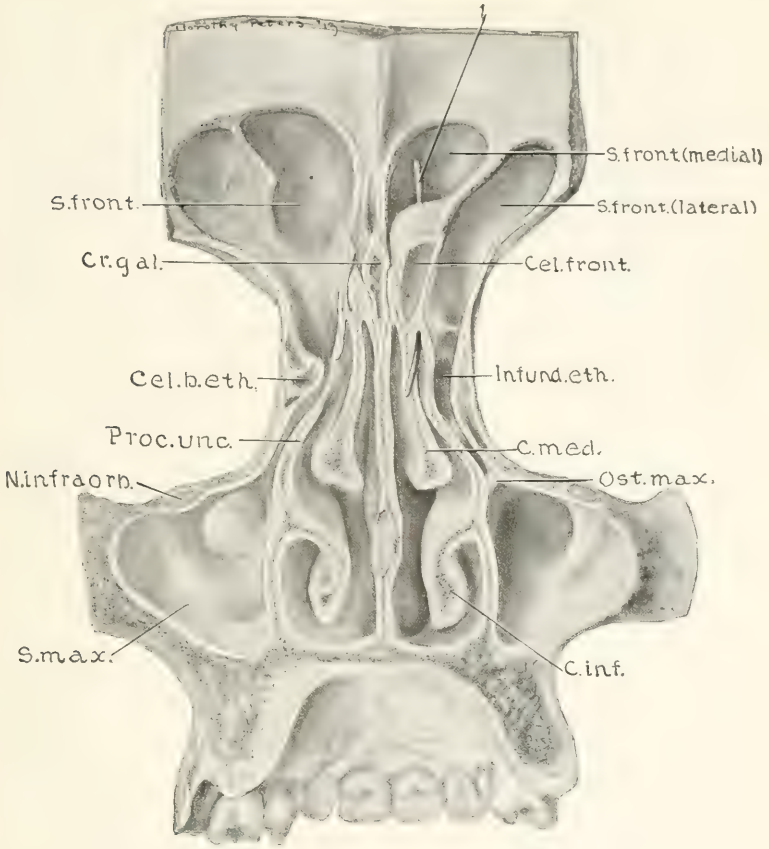

Fig. 215.—Specimen twenty years and five months old. (Series D, No. 90.) Posterior view of a coronal section 18 mm. posterior to the nasion. The posterior walls of the sinus have been removed to show the relations of the sinuses and their ostia. Note that on the right side there are two distinct sinus frontales present. The more medial of these two sinuses developed from a cellula frontalis, medial to the processus uncinatus, while the lateral one developed from the infundibulum: *1*, Probe through the ostium of the medial sinus frontalis, the ostium being anterior to the cellula frontalis shown in illustration; *S.front.*, sinus frontalis; *Cel.front.*, cellula frontalis; *Infund.eth.*, infundibulum ethmoidale; *C.med.*, concha media; *Ost.max.*, ostium maxillare; *C.inf.*, concha inferior; *S.max.*, sinus maxillaris; *N.infraorb.*, nervus infra-orbitalis; *Proc.unc.*, processus uncinatus; *B.eth.*, bulla ethmoidalis (extreme anterior portion); *Cr.gal.*, crista galli. (From Davis, "Development and Anatomy of the Nasal Accessory Sinuses in Man.")

probability of such infections being the primary source for metastasis.

A consideration of the nasal accessory sinuses as a source of metastasis will unmistakably establish:

First, that the peculiar intricate anatomic build of these structures, together with their particularly exposed location and intimate relation to one another, render them particularly vulnerable to infective invasion.

Second, that their peculiar defenseless condition and tolerance to infection and their hidden positions cause them to become seats of unsuspected infective processes, both locally and generally.

Third, that abundant clinical evidence is at hand to warrant the statement that these several structures, individually and collectively, are a very frequent source or focal center of general systemic infections.

Before we consider the pathologic processes of the nasal accessory sinuses and their metastasis, let us review briefly, with the aid of these charts, the anatomy and relation of these structures.

The **maxillary sinus** may be likened to a pyramid, with the lateral wall of the nose forming the base; above is the orbital plate of the superior maxillary; *anteriorly*, the canine fossa; *posteriorly*, the pterygomaxillary fossa. Average dimensions are: height, $1\frac{1}{2}$ inches; breadth, 1 inch; depth, $1\frac{1}{4}$ inches; average capacity, 1 ounce.

In the newborn the antrum is present, although very small, about the size of a small bean. It occupies a space internal to and not below the orbit. The subsequent growth occurs through downward development of the alveolar process and lateral absorption of cancellous bone. It does not assume its permanent shape until after the eruption of the permanent teeth, with complete development of the upper jaw at the fifteenth to the eighteenth year. Normally, it has *one* ostium, which opens into the nose beneath the anterior superior portion of the middle turbinate. Sometimes there are several accessory openings. The roof of the antrum also forms the *floor* of the orbit; the infra-orbital nerve also runs partly in a channel in it; the floor of the antrum is in close relation with the teeth, particularly the second bicuspid and first molar. The external wall is in relation with the face, the internal wall with the nasal cavity.

The Ethmoids.—The ethmoid labyrinth is situated at the anterior part of the base of the cranium, between the two orbits, at the root of the nose. It consists of a horizontal or cribriform plate, a *perpendicular* plate which forms part of the *septum* and two lateral masses of cells. The *cribriform plate* forms part of the anterior fossa of the base of the skull. The *crista galli* projects upward from the plate; on either side of the crista the *olfactory bulb* rests. The olfactory nerves pass through foramina in the cribriform plate, and are enveloped in a prolongation of dura mater which connects more or less intimately with the nasal mucosa.

This anatomic formation favors the ready transmission of infection from the nasal cavities to the meninges. The *number* of *cells* in each lateral mass varies from 2 to 12. Each cell has its separate outlet. Roughly they are divided into *anterior middle* and *posterior*. The anterior cells empty into the hiatus semilunaris and into the middle nasal passage; the *middle* and *posterior* empty into the superior nasal passage. The *outer wall* of each mass is formed by the *os planum* or *lamina papyraceus*, which also forms a considerable part of the *inner wall* of the orbit. Three important structures pertain to the ethmoids. (1) The *processus uncinatus;* (2) the *hiatus semilunaris;* (3) the *bulla ethmoidalis*. These structures are all in the *middle meatus* and are covered by the anterior end of the middle turbinate, which must be removed or shoved aside to disclose them. The *processus uncinatus* is a thin and narrow process of bone; behind it lies a trench or furrow, which is the hiatus; the hiatus is a shallow curved furrow or depression, situated in the middle nasal passage, between the uncinate process and the bulla, and in normal cases is covered by the anterior third of the middle turbinate. Its depth or bottom is known as the *infundibulum;* the frontal, anterior ethmoidalis, and antrum drain into the hiatus. Back of this is a bulbous prominence, "the bulla," the largest and most prominent of the ethmoid cells. The *relations* of the ethmoids are important. *Above* is the brain, *externally*, the eye, *below*, the nose and antrum; *behind*, the sphenoid.

Frontal Sinuses.—Extend horizontally outward 1½ to 2 inches

from the median line. A partition separates them; sometimes more than one is present. Each has an opening or duct into the nasal cavity, emptying into the *middle meatus*. The *anterior* wall is formed by the frontal bone, the *posterior* wall forms part of the wall of the anterior cranial fossa and is in contact with the frontal lobe of the brain. The *inferior* wall forms part of the roof of the orbit and is the *thinnest* portion.

The *nasofrontal* duct of the frontal sinus is usually about $\frac{1}{4}$ inch in length; it is really a part of the *hiatus semilunaris* into which it leads and with which it is continuous; its direction into the nose is *downward*, *forward*, and slightly *inward;* it is about 3 mm. in diameter.

The Sphenoid Sinuses.—These are hollowed out of the body of the sphenoid bone, are situated directly behind the ethmoid capsule at the *posterior* and *superior* portion of the nasal cavity. The ostium opens into the superior nasal passage on the superior wall of the sinus. The *superior* wall of the sinus is in direct contact with most important intracranial structures, the *optic nerve* and *optic chiasm*, the *cavernous sinus* and *pituitary* body in the *sella turcica*. The *external* wall helps to form a portion of the middle cerebral fossa and is in direct communication with the *cavernous sinuses* and internal carotid artery. The *optic nerve* and a part of the optic commissure generally lie upon the roof. The *cavernous* sinus run along the upper part of the *external wall*. The *internal* carotid artery lies in a groove directly against the posterior and superior angle of the sinus. The *gasserian ganglion* lies external to the cavernous sinus. The nasal spaces, including each meatus and all of the accessory sinuses is lined with ciliated epithelium, the *motion wave* of the cilia being directed toward the ostium of the sinus.

The blood supply of nasal fossa is rich and the branches anastomosis freely. *Arteries:* The sphenopalatine branch of the *internal maxillary* (one of the terminal branches of the external carotid) is the chief artery. It enters the cavity by the sphenopalatine foramen and supplies meatuses, turbinates, septum, etc.

The anterior and posterior ethmoid branches of the oph-

thalmic artery also supply portions of inferior turbinates, meatus, and septum.

Veins.—The veins form a dense plexus in the mucous membrane, those in the deeper parts of the membrane being especially large and closely arranged so as to approach the structure of a cavernous tissue. This is exhibited over the whole of *lower turbinal*, lower and posterior portion of middle, and posterior end of superior turbinals. The trunks leave the cavity with arteries. The sphenopalatine veins empty into the pterygoid plexus. The *ethmoid* veins join the ophthalmic and the veins of the dura mater; small veins also pass out at the margin of the nares to join the venous plexus of the upper lip; some also pierce the nasal and superior maxillary bones to join the facial veins.

The *lymphatics* of the *nasal fossæ* are numerous. They form a close plexus in the mucous membrane; communicate with the lymphatic spaces which enclose the branches of the olfactory nerve, and these in turn communicate with the subdural and subarachnoid spaces of the cranium.

Bacteriology.—The micro-organisms found in sinus disease are in their relative order of frequency:

(1) The influenza bacillus;
(2) The Diplococcus pneumoniæ;
(3) The Staphylococcus pyogenes;
(4) The streptococcus;
(5) The Bacillus coli;
(6) The pseudodiphtheria bacillus.

The **function** of the sinuses is still unsettled. Different observers assign to them the following rôles: Adjuncts to inspiration, moistening the inspired air, adjuncts to olfaction and lightening the bones of the skull.

Frequency.—Sinus infections are perhaps more common than is generally supposed. Different observers from postmortem examinations, in deaths from all causes, give a percentage of sinus involvement ranging from 22 to 43 per cent. Diseases commonly producing sinus infection during some part of their course are influenza, pneumonia, diphtheria, erysipelas, scarlet fever, measles, small-pox, tuberculosis, and typhoid fever.

It is not uncommon for these diseases to have associated with them other infections, such as the joint infections met with during pneumonia, typhoid, or influenza. Possibly the susceptibility of the sinuses to these diseases and the later joint and other systemic complications met with may have a closer relationship than we usually suspect. Acute sinus infection is frequently the cause of serious complications, involving the pleura, lungs, heart, and kidneys.

Complications—Neighboring Structures.—(1) *Eye and Orbit.*—Infections of these structures, secondary to sinus involvement, are frequent, and may be orbital cellulitis, orbital abscess, affections of the conjunctiva, cornea, uveal tract, retina, retrobulbar neuritis, thrombosis of the vena centralis, paralysis of accommodation or mobility of globe, etc. Blindness may result from atrophy, consequent upon an optic neuritis or secondary to a thrombosis of the central vein of the retina. From a surgical point of view, it is not improbable that many of the obscure postoperative infections, such as those arising occasionally following cataract extraction, may be explained on the theory that the pathologic condition is a metastatic process, having its origin in the sinuses, the tonsils, or a disease of the teeth, such as pyorrhea alveolaris.

Certainly, disclosures, which modern investigation supplies, of the protean rôle metastatic processes exhibit, suggest that it would be wise, in at least all major ophthalmic surgery, to adopt, as a routine measure, the investigation of the sinuses, tonsils, and teeth before the operation is undertaken.

(2) *Cerebral Complications.*—These are quite frequent: various forms of meningitis, extradural and intradural, cerebral abscess and thrombophlebitis of the venous sinuses occur.

Systemic.—The literature abounds with repeated references to the probable association of sinus disease to systemic infections.

Darling (from the Pathologic Laboratory of the Ancon Hospital, Isthmus of Panama), in an investigation conducted to determine the relation of inflammation of the accessory nasal sinuses to pneumococcus infection, made autopsies on 52 cases. His conclusions were that 92 per cent. of all pneumococcus infec-

tions coming to autopsy show in a very marked degree more or less typical pneumococcus inflammation of one or more of the accessory nasal sinuses; 91 per cent. of the lobar pneumonia cases showed a sinusitis. All his cases of acute pneumococcus meningitis presented an inflammation of one or more of the sinuses. He believes that the port of entry of the pneumococcus is in most instances an accessory nasal sinus—the mucous membrane of which is probably fitted for infection by the pneumococcus by an antecedent influenza or rhinitis.

It is, to say the least, as Holmes points out, a suggestive coincidence that two pathogenic bacteria, the pneumococcus which produces such diseases as lobar pneumonia, acute purulent cerebrospinal meningitis (pneumococcus), endocarditis, pericarditis, etc., and the streptococcus, which produces meningitis, erysipelas, cerebral and metastatic abscesses of various kinds, and also ear infections have their normal habitat in the nose and accessory sinuses.

Erysipelas.—In many cases, it seems clear that erysipelas is secondary to an infection of an accessory sinus. Authorities differ in assigning to the erysipelas the primary or secondary rôle. Enough evidence, however, is obtainable to suggest that in all cases the sinuses should be investigated as the possible source of the infection.

Gastric Disturbances.—These are common in connection with sinus affections. Nausea, vomiting, loss of appetite, etc., probably result from the constant swallowing of the purulent secretion with reabsorption of ptomains.

Endocarditis and Nephritis.—These are quite commonly a sequence of sinus infection.

Joint Infections.—The association of joint infection to sinus infection is frequent. While the accessory sinus has not been so specifically associated with these involvements as have other structures of the upper respiratory tract, notably the tonsil, it is not unreasonable to assume that a careful investigation will support the theory that many obscure lesions of the joints may find their focus of infection in the accessory sinuses of the nose.

General.—There are many cases of sinus infection exhibiting

symptoms simulating other diseases, such as malaria, nervous and mental disorders, etc. The patients may have increased temperature, chills, general lassitude, depression, inability to concentrate, melancholia, aching in the joints, headache, etc. Such patients often are the victims of sinus infection.

Nasal Reflexes.—Under this head I will mention only the well-known experience that many cases of asthma disappear when a sinus or some nasal difficulty is remedied.

Symptomatology.—Under the head of symptomatology I will not discuss in detail the symptoms of sinus infection, but, I would like to emphasize a variety of symptoms common to such infections with which I meet very often.

The patient complains of "depression," "lack of concentration," "inability to carry on a sustained effort," "irritation," "sensitiveness," etc.

This "symptom complex" is, in my experience, frequently encountered. The relief and the mental change which these patients exhibit after operation are astonishing. These cases are often considered hypochondriacs. Their complaints are considered imaginary because their descriptions of their sufferings are so vague, so complex, and so fantastic. Often, since they are denied serious attention by the physician, they drift into the hands of the Christian scientist, the fakir, and the exploiters of other cults. Their sufferings are *real*, not imaginary, not hysterical, and the relief they obtain is almost marvelous. I do not hesitate to say that results from procedures in any department of medicine do not surpass those attained by proper attention in cases of infection of the sinuses.

CLINIC OF DR. J. C. FRIEDMAN

MICHAEL REESE HOSPITAL

CHRONIC PAIN IN THE RIGHT ILIAC FOSSA

How Can One Diagnose Chronic Appendicitis? Aschoff's Theory. History of Acute Attacks of Sharp Colicky Pain with Absolutely Free Intervals Most Typical.

Evidence of Chronic Appendiceal Inflammation Found Postmortem in 75 Per Cent. of Adult Cases. Deep Tenderness at McBurney's Point Common in Healthy Persons.

Spastic Constipation, Dyschesia, and Visceroptosis—Their Treatment.

Report of 14 Cases Observed Before and After Appendectomy.

THIS young woman is twenty-five years of age, unmarried, and entered the hospital three weeks ago with the following history: Two weeks ago she was seized with a sudden, severe pain in the right iliac fossa, colicky in nature, not radiating, fairly constant, but varying in intensity from time to time. The skin over this area was tender, and the attack was accompanied by nausea and vomiting, though pain was not increased by food. She has felt feverish at intervals since that time. There has been marked loss of appetite and considerable loss in weight, the exact amount of which she does not know. She has had frontal headaches for one week.

Previous history: Had a similar attack six weeks ago, which lasted three days, and another attack one year ago. Had measles, scarlet fever, and diphtheria in childhood, pneumonia six years ago, and typhoid four years ago. Has been constipated for the past five or six years, ever since she started to do office work. Her menstrual periods are regular and bear no relation to her pain. Family history is negative.

On examination we find an extremely slender young woman, rather poorly nourished. The head and throat are normal, heart and lungs negative. The abdomen is narrow and flat. The distance between the lowest rib and the crest of the ilium is very small. Liver, spleen, and kidneys are not palpable. There is marked tenderness and resistance in the right iliac fossa, not, however, strictly localized at McBurney's point. The skin in this region and in the right lumbar region posteriorly is hyperesthetic. The cecum and ascending colon are easily palpable, not indurated, and somewhat tender. The reflexes are normal. The temperature during the first ten days of her stay in the hospital ranged from 98° F. in the morning to 99° and 99.2° F. in the afternoon. The stools obtained after an enema were soft, showed no increase in mucus, and no blood. The white count varied between 9000 and 11,000; differential count normal; hemoglobin, 85 per cent. Rectal examination was negative. A bismuth meal was given and the picture showed an orthotonic stomach, the colon in normal position, normally movable. The appendix was not seen. The tenderness complained of seemed to be about the point of the ileocecal valve rather than at the base of the appendix.

To sum the matter up, then: We have a young woman who, until a year ago, was in fairly good health except for severe constipation, and during the last year has had three fairly distinct attacks of pain localized in the right iliac fossa, accompanied by nausea and vomiting and, she thinks, some fever. A subfebrile temperature has been found during her stay at the hospital. On examination nothing abnormal is found except some tenderness in the right iliac fossa, which fluoroscopy shows to be about the point of the ileocecal junction.

Two facts stand out: First, three distinct attacks of pain and fever; second, constipation. Our first thought would therefore be either a chronic inflammatory process in the right iliac fossa with accompanying constipation, or chronic constipation with recurring attacks of colitis. Of the former condition, chronic simple appendicitis comes to our mind first; there is no good reason for thinking of a tubercular process here, and

the possibility of an inflammatory process in the internal genitalia is not borne out by rectal examination nor by the absence of painful menstruation. The *x*-ray, it will be remembered, showed no adhesions around the cecum. The pain continued during her first week's stay in the hospital, and as no further progress was made between the diagnoses of chronic appendicitis and constipation, an operation was decided upon and performed by Dr. Greensfelder. The findings were quite negative. The appendix was perfectly free, of normal size and consistency, and no abnormality was discovered in this region. The microscopic examination by Dr. Kirk disclosed a moderate fibrosis, certainly not enough to account for her symptoms. Since the operation the patient has felt perfectly well, perhaps from the fact that her bowels are moving regularly. The diagnosis at the present time is *not* chronic appendicitis, but spastic constipation, with attacks of either slight inflammation or colic of the large intestine. That the above is not an isolated instance any internist or surgeon who is sufficiently interested to follow his cases for at least one year after appendicitis operations can testify.

Before discussing the matter further I wish to present another case to you: This young woman is unmarried, nineteen years of age, a coat-finisher by occupation. She entered the hospital six days ago, complaining of epigastric distress immediately after eating, independent of quantity and quality of food, nausea but no vomiting, some anorexia and loss in weight, some pain in the interscapular region and sometimes in the right lumbar region (the latter pain especially is worse on walking or at the end of a day's work), frequent headaches, marked constipation.

Previous history: Has had pains similar to the present for the past four years. Two years ago was operated for catarrhal appendicitis and displaced kidney, with no relief of symptoms. Family history is negative as to tuberculosis. Menstrual history: No abnormality, especially no increase in the above-mentioned pains during menstruation.

Examination.—Poorly nourished, anemic girl. Eyes normal. Throat normal. Lungs and heart show no abnormality. Ab-

domen flat, scar in lower half of right rectus border, musculature poor; spleen palpable; right kidney movable (second degree). There is marked tenderness on deep pressure in the right iliac fossa, fairly well limited to McBurney's point, viz., the junction of the middle and outer thirds of the spino-umbilical line. Deep reflexes are very active. Pharyngeal reflex absent. No sensory disturbance. The urine is normal. Blood examination shows hemoglobin 72 per cent., white and red counts normal. Her temperature during her present stay in the hospital has been normal. Examination of stomach contents showed no abnormalities. Stools normal, no blood or mucus. *X*-ray examination of gastro-intestinal tract shows marked ptosis of stomach, the lesser curvature being below the iliac crests and the transverse colon correspondingly low; the cecum was fairly movable, and the point of deep tenderness again corresponded to the ileocecal junction.

To sum up then we have a poorly built and poorly nourished young woman with epigastric pain occurring immediately after taking food, nausea and marked constipation. Examination reveals normal lungs and heart, deep tenderness in right iliac fossa, and marked general visceroptosis. As to the diagnosis, we can rule out a primary stomach disturbance, such as ulcer, while gallstones, kidney stones, etc., are equally improbable. We have left then the appendix, visceroptosis, and constipation. The appendix was removed two years ago without relief, and so, by exclusion, we are left with the syndrome of visceroptosis and constipation, which is the final diagnosis.

The common feature in these two cases is that both were operated on for chronic appendicitis, one with relief and one without, but neither appendix disclosing enough pathology to justify the operation. I have been following a number of cases of so-called chronic appendicitis during the last two years and have collected 14 similar cases of patients who have come under observation before or after appendectomies, in the former group the operation disclosing insufficient pathology to account for the symptoms, and in the latter group the symptoms persisting after the operation.

Two questions are raised by these facts: First, how common is chronic appendicitis and how can one diagnose it? Second, where it is falsely diagnosed, what is the pain due to? It is a very old question and has been discussed during the past fifteen years by clinicians all over the world. From a pathologic standpoint the best answer has been given by Aschoff, who believes that there is no such thing as a gradually progressing chronic process in the appendix, but only acute exacerbations of an incompletely healed acute process. Clinically this may be expressed in the following terms: Unless one can get a history of one or more definite fairly typical acute attacks with free intervals the diagnosis of chronic appendicitis will probably be wrong. To illustrate this point, I wish to read you the following history: Mrs. M., married, twenty-seven years of age, was suddenly taken ill two days ago with cramp-like pains in the abdomen, at first generalized, later localized in the right iliac fossa, sometimes radiating to the back on the right side, with vomiting. She had a similar attack ten months ago and a year ago. Menstruation normal and has no relation to pains. Examination shows deep tenderness and slight rigidity at McBurney's point, and *x*-ray examination reveals the tender point *below* the ileocecal valve, *i. e.*, at the base of the appendix. The stomach is prolapsed. White blood-count 10,000 to 11,000, temperature 99.6° F. maximum. At operation a thickened subacutely inflamed appendix was found. This history resembles that of the first patient demonstrated, in that there were three distinct attacks, each with slight temperature and localized tenderness, but differs from it in the typical colicky character and evident severity of the pain and in the complete freedom of symptoms in the intervals, and in the *x*-ray findings of the tender point at the base of the appendix and not at the ileocecal valve. I think few men would hesitate, and properly so, to diagnose recurrent appendicitis in the last case. I also think that many physicians would advise operation in the first, but they would be wrong in a large percentage of such cases, even if evidences of old appendicitis were found at operation. To quote Aschoff again, "Such evidences of old inflammatory processes are found postmortem in 75 per cent. of all

adult subjects," while Codman, of Boston, in 100 cases of abdominal section for other reasons, found evidences of previous appendiceal inflammation in 71. Therefore, if we open the abdomen of any adult patient the chances are about three to four that we can find enough pathology in the appendix region to claim that he was suffering from chronic appendicitis. To demonstrate this point I present the following case: Miss T., unmarried, twenty years of age, complained for last two months of pain in right lower quadrant, of rather sudden onset, but not continuous; not severe enough to keep her awake at night; no relation to food taking or menstruation; accompanied by nausea and vomiting and she thinks by chills and fever; extremely constipated. She admitted having been under a terrific mental strain since the beginning of the European war. The points of importance in the examination are the tenderness over the whole right side of the abdomen, most marked in right lower quadrant, with some rigidity. Pharyngeal reflex absent. Temperature normal. *X*-ray reveals the point of greatest tenderness over the ileocecal valve. The diagnosis of neurasthenia was made, which was *not* concurred in by the surgeon, who operated and found an appendix free from adhesions but with a stricture in the middle and a dilated distal half, and no evidences of a recent acute inflammatory process. It is this last point which made me adhere to the original diagnosis of neurasthenia even after the finding of a pathologic appendix, for I believe (with Aschoff) that no chronic processes except broad, strong bands of periappendicular adhesions can cause symptoms, all other true appendicitis being acute exacerbations. The other piece of evidence generally offered, viz., that the patient has been free from symptoms since the operation must be cautiously accepted. For instance, Kuttner, in an article written thirteen years ago on this question, mentions a blacksmith, thirty years of age, operated on five years previously for so-called appendicitis, in whom the appendix appeared normal and was *not removed!* Since that time not a symptom has recurred. It is especially in neurasthenic subjects of marked suggestibility that this type of cure occurs. However, cure does not occur nearly so often as

the average physician thinks. For instance, Melchior and Loser report permanent cure in 95 per cent. of cases of real chronic appendicitis, while in cases without typical attacks only 60 per cent. were cured.

The diagnosis then is important. What are the main points? The first is the history, especially of typical acute attacks, with free intervals. I have already spoken of this point. Constipation from appendicitis is the exception and not the rule, as is generally believed. For instance, Codman found constipation in 21 out of 61 cases. On examination, deep tenderness and even rigidity on palpation are utterly unreliable. Keith some fifteen years ago found by dissection of fifty subjects that McBurney's point lay directly in front of the ileocecal valve and about 1 inch *above* the base of the appendix. Our radiographer, Dr. Turley, has paid particular attention to this point and comes to the same conclusion, viz., that the base of the appendix in the majority of cases does not lie directly beneath McBurney's point.

The two accompanying plates illustrate this fact. Figure 216 is a colon in its normal position at the level of the crest of the ilium with the spino-umbilical line in which McBurney's point is situated above the base of the appendix (x). Figure 217 is the most frequent variation in position of the colon, viz., a ptosis, with the appendix filled with bismuth, showing its base still further removed than normal from the spino-umbilical line.

Another fact, better known than the former, is the extreme frequency with which tenderness on fairly deep pressure is elicited in the normal individual. Treves tested 27 healthy medical students and found tenderness there in 24 of them. He believes it is due, first, to the ileocecal junction sensitiveness, and, second, to the fact that the eleventh dorsal nerve enters the rectus sheath at this point. Tenderness, then, at McBurney's point, as an aid in the diagnosis of chronic appendicitis, is a sign of doubtful value as compared with its importance in acute appendicitis. Other signs are equally misleading; for instance, inflating the colon with air, when, in a supposedly inflamed appendix, the tenderness over McBurney's point will be in-

creased. This is probably true in some cases, but it is also true that in some of the conditions most difficult to distinguish from

Fig. 216.—Röntgenogram showing relation of base of appendix (*x*) to spino-umbilical line in average build.

chronic appendicitis (chronic constipation, colitis, etc.) the same increase in tenderness occurs.

A much more helpful method is fluoroscopic examination of the bismuth-filled colon. I cannot help but doubt the reported

frequency of pylorospasm due to chronic appendicitis. Pictures of the bismuth-filled appendix have not been of value to us. The two findings in my experience which have been of positive

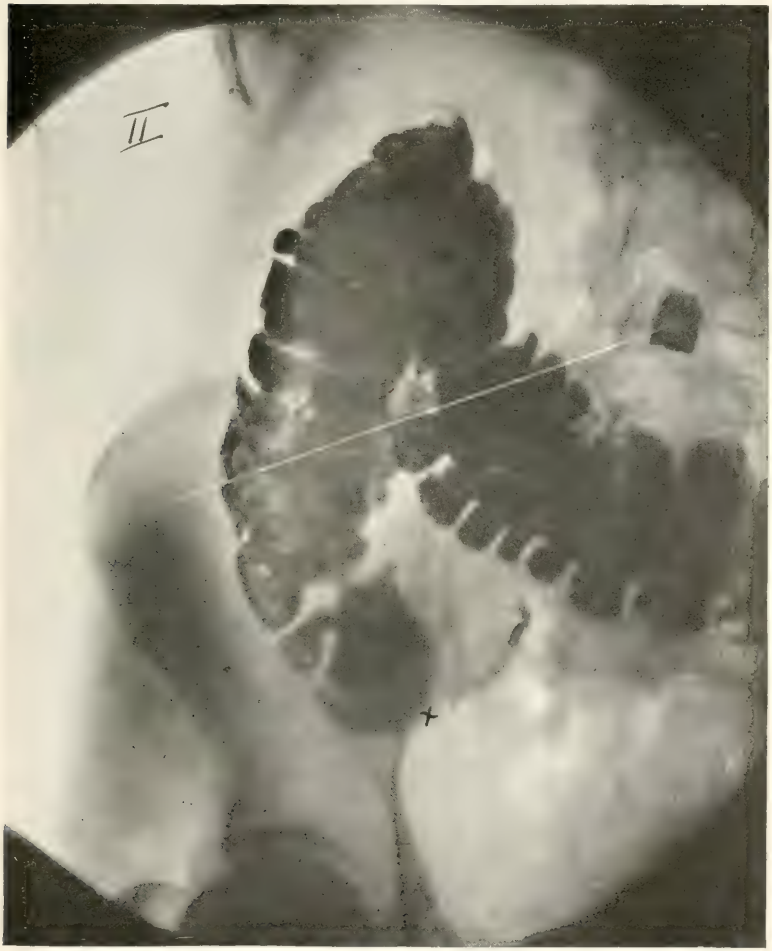

Fig. 217.—Röntgenogram showing relation of base of appendix (*x*) to spino-umbilcal line in enteroptotic build.

value are identifying the tender point as really lying above the base of the appendix and demonstrating a cecum fixed by adhesions. On the other hand, Wilm's movable cecum is probably

of no greater pathologic significance than the various veils and membranes which have been shown to be normal congenital growths. To sum up, we are safe in diagnosing chronic appendicitis if we get a history of typical attacks with free intervals, and if we can with the *x*-ray demonstrate a tender appendix or adhesions. But if we take the other group of cases, with long-continued pain in neurasthenic individuals with constipation and so forth, we shall have no results from appendectomy in about one-half the number (to take the conservative statistics of Melchior and Loser), and it is of the diagnosis and treatment of this class of cases, as typified by the first and second patients presented this morning, that I shall speak for the rest of the period.

First of all, we must rule out by the usual methods the ordinary diseases from which subacute and chronic appendicitis must be differentiated, viz., stone in the ureter or kidney, ulcer of the stomach or duodenum, carcinoma, or tuberculosis of the ileocecal region, chronic lead-poisoning, inflammation of the appendages in women, and mucous colitis. Differentiation between gallstones, ulcer of the duodenum, and appendicitis is often difficult, owing to the fact that they all in their atypical forms may give rise to that feeling of discomfort after eating which we call indigestion. Prolonged observation, repeated gastric analyses, both as to chemistry, and especially motility, are often necessary before we can come to any conclusion. If, however, we have ruled them out, what conditions are left to differentiate from? In the 14 cases above mentioned one or more of the following findings were present: Chronic constipation with or without mucous colitis, and neurasthenia and visceroptosis either alone or in combination. To demonstrate their presence is one thing, however; to determine that they are giving rise to the symptoms complained of is more difficult. To save time I shall simply state my own experience in this connection: Visceroptosis causes two symptoms, *i. e.*, a feeling of distress, sometimes of actual pain, although dull, in the epigastrium, immediately after taking food of any kind, which pain does not radiate and is often relieved by lying down, and a similar feel-

ing of discomfort or pain in the right iliac fossa and in the lumbar region posteriorly, on walking or working, which is also relieved by lying down. Fluoroscopic examination in such cases reveals a visceroptosis with atony of the stomach and often a six-hour residue, while pressure upward by the hand on the lower half of the abdomen gives the patient relief; the tenderness found in the right iliac fossa is above the appendix and the cecum is freely movable. Such a history and such findings would justify us in concluding that the visceroptosis was causing at least some of the symptoms.

Types of Constipation.—We may for convenience divide constipation into two types: The one due to slow passage of feces through the intestines and the other due to inability to defecate completely. The former we again subdivide into ordinary atonic constipation, due to weakness of the musculature and diminished reflex activity, which is the most frequent type, but does not concern us here, and spastic constipation, of which Case No. I is an example. This type of colonic slowing is probably the result of spasm induced reflexly by the presence of some irritant in the colon of a patient with an abnormally excitable nervous system. The nervous element is shown by the neurotic temperament of all patients so afflicted, and the frequent history of worry or overwork immediately preceding an attack. The irritant which excites the exaggerated reflex is probably in most cases hardened feces, the retention of which is due to the ordinary neurasthenic constipation generally present in the intervals between attacks. As a result of these spasms the cecum becomes overfilled, atonic and tender, and, as in our case, closely simulates an inflamed appendix. Thus, in 13 cases of spastic constipation mentioned in one article on the subject, 11 had had appendectomies performed. In the other main division of constipation, the so-called *dyschezia*, there is generally a normally rapid movement through the colon, but the act of defecation does not empty the descending colon completely, and, as a consequence, we find on rectal examination a considerable mass of feces even after defecation. This type concerns us only because it frequently accompanies visceroptosis, as in Case No. II, being due

partly to the same cause as the visceroptosis, viz., weak abdominal muscles.

The third cause of pain in the right iliac fossa is of nervous origin, *i. e.*, neurasthenia, either alone or more frequently combined with one or both of the other factors. Its combination with the other two causes is clear. Visceroptosis is apt to give rise to uncomfortable sensations, especially in neurasthenics, and, as explained above, spastic constipation is practically only found in patients with abnormally excitable nervous systems. But why should neurasthenics be prone to localize their pain in the right iliac fossa even when visceroptosis and constipation are absent? It is explained, I think, by the widespread knowledge and fear of appendicitis. This was well illustrated in a patient I saw recently, who had his first attack on the day one of his friends was operated upon and died from an acute appendicitis. We are all acquainted with neurasthenic families in which if one member is operated on for appendicitis we are certain shortly after to see some of the others with indefinite pains, who wish to be assured of their freedom from the dread disease.

Treatment.—If then, after careful consideration, we have made the diagnosis of pseudo-appendicitis, *i. e.*, one or more of the above triad, we have still a difficult problem, namely, the treatment. Of course, no operation is indicated. Fixing kidneys is out of date, removing appendices does not in itself cure constipation, and sidetracking colons for stasis is a delusion and a snare. There remains then the medical treatment.

The treatment for visceroptosis consists first in strengthening the abdominal muscles, which may be accomplished by the various Swedish movements designed for that purpose, but in the young, gymnasium work suited to their strength is far better. The stimulation and improved general health reacts favorably on the accompanying neurasthenia. Nothing adds more to the comfort of such patients than a properly made abdominal support. The important features of such a binder are that it be broad enough to include the length of the abdomen and that it lace in front so that it may be applied by the patient while lying down with the hips elevated on a pillow. It is laced

tightly below and more loosely above. An attempt to increase the abdominal fat by extra nourishment, such as milk and cream mixtures between meals, is also of value.

Spastic constipation deserves special consideration. To treat these cases by prescribing a coarse diet and cathartics simply increases the spasm. It is with such patients that a non-irritating diet, one with the smallest possible residue, is most successful. This consists of thoroughly cooked softened food; temporarily at least all meats are passed through a meat chopper, all vegetables and fruits are passed through a hair sieve. In this way, the connective tissue and cellulose are either removed or so finely comminuted as to be non-irritating to the large intestine. A further means of relieving spasm is by the use of atropin, $\frac{1}{200}$ grain one or more times a day, gradually increasing the dose until the physiologic limit is reached. The underlying atonic constipation is best corrected by 6 or 8 ounces of cotton-seed oil injected into the rectum at night and retained until morning, the number of injections being gradually reduced. It is rarely necessary, even at first, to give small divided doses of cascara as an adjuvant. It is unnecessary to go into the treatment of the accompanying neurasthenia. Freudian psychanalytic methods are quite unnecessary in the majority of cases, and a straightforward psychotherapy suited to the intellectual status of the patient is quite sufficient.

CLINIC OF DR. CHARLES SPENCER WILLIAMSON

COOK COUNTY HOSPITAL

HANOT'S DISEASE. BILIARY HYPERTROPHIC CIRRHOSIS OF THE LIVER

A Symmetrically Enlarged, Hardened Liver, with an Enlarged Spleen, Fluctuating Jaundice, Absence of Ascites, and Irregular Temperature Constitute the Typical Symptom-complex of this Rare Disease. Classification and Differential Diagnosis.

I AM showing you this patient because of the relative rarity of his disease. The man is a Russian, thirty-three years of age, a porter by occupation, who came into the hospital about three months ago with the following history:

He was brought to the hospital in an ambulance, and was complaining of a *dull persistent aching in the right* hypochondriac region, which was not influenced by anything he had tried. He was rather deeply jaundiced, had marked thirst, and a diffuse itching of the skin. Thus, the complaints were four: aching, jaundice, thirst, and itching. The patient states that he was perfectly well up to three weeks before coming into the hospital. He then noticed that the whites of his eyes were becoming yellow. The jaundice has been persistent since the onset. When he entered the hospital he was about the color of a pumpkin. He said he felt pretty well, but came to the hospital on the advice of his physician. He denies all infections, particularly venereal infection; he has been a total abstainer for several years, and previous to that he used alcohol to only a slight degree. From the statements of friends we have reason to believe that this is correct. The bowels are regular, and the color and consistency of the stools have been pretty nearly normal all the way through. The stools have never been acholic. He has no urinary symptoms. His weight has remained sta-

tionary. He had rheumatism many years ago, but no other sickness. He knows nothing about his family history.

Physical examination when he came in revealed a well-developed young adult, in good nutrition, rather plump than otherwise, and pretty deeply jaundiced. He has lost neither weight nor strength, and feels comparatively well. His temperature on admission might be called subfebrile; he was running a temperature of 99° F. and a fraction, and that has been about the course ever since, except that it has been normal now for some little time.

Examination of the chest was negative; the first abnormal findings were detected in the abdomen. It was and still is greatly distended, and, as we examine him more closely, we see that this distention is largely in the upper part. Both hypochondria protrude, particularly on the right side. This has not changed since he came in the hospital.

On palpation, we find the abdomen pretty soft at the present time, and on the right side we find a big mass which comes down to within a finger's breadth of the umbilicus in the nipple line. In the middle line it comes to about two fingers above the navel, and extends to the left until it reaches the costal arch in the left nipple line. Above it reaches as high as the lower margin of the fifth rib; that is, absolute flatness is that high; relative dulness reaches to the nipple. So we have an rea of flatness about the width of nine or ten fingers. The edge is slightly rounded, not nearly so sharp as is ordinarily the case. It is hard, and we can trace the edge with great ease. The surface is smooth and no irregularities can be felt.

Palpating the Liver.—Now let me say a word about the proper way to palpate a liver: Have the patient draw the knees up; sit down on the right side of the patient, put your left hand on the patient's back, with the fingers just below the twelfth rib. Then place the fingers of the right hand below the liver in front, making moderate pressure and holding steadily. Then have the patient take a deep breath, and you will feel the edge of the liver slip over the index-finger of the right hand. On careful palpation we find nothing on the surface—no bumps, no de-

pressions, no indentations on the edge except the one between the right and left lobes. At no time *have we found any evidence of ascites* or portal obstruction.

I will now show you the correct way to examine the spleen. Place the patient in a position half over on his right side, put your left hand and fingers in behind and below the last rib, pushing it forward, while the right hand feels for the edge of the spleen in front. In this case the spleen comes a full four fingers' breadth below the edge of the ribs and as far upward as the seventh rib. Bearing in mind the normal position of the spleen, you will see that here it is elongated considerably and greatly increased in the transverse diameter. The blood shows nothing special. There is no evidence of increased fragility. The urine gives a positive bile reaction.

Differential Diagnosis.—The first question is as to the differential diagnosis. What sort of a liver have we? An important point I want to call to your attention is, that it is *symmetrically enlarged*. The edge is hard and not quite so sharp as an ordinary liver would be. *Whenever you find the edge of a liver slightly rounded, hardened, and thickened you may be sure there is some diffuse interstitial process present.* Let us say, for example, that we have tumor masses in the form of metastases in the liver. These often leave the liver edge unchanged. You will find much enlargement, but the edge is still sharp and fairly soft. If we have some sclerotic process it leaves the edge hard and round, and whenever you find the edge of the liver harder and rounder than normal it is the best evidence that you have some sort of cirrhotic change in the liver. The edge of this liver and its hardness makes it perfectly clear that we have some diffuse interstitial process; that is, some type of cirrhosis. It is not a tumor, such as a carcinoma or an echinococcus cyst.

We have a number of types of cirrhosis; the common types in the approximate order of their frequency are:

First. Alcoholic cirrhosis.

Second. The so-called "fatty cirrhosis," which is a fatty infiltration of the liver cells in addition to an alcoholic cirrhosis.

Third. Luetic cirrhosis, which is very characteristic; it is a tertiary lesion of syphilis. It occurs invariably as the result of multiple gummata, which heal and leave scars. The final result is called the "hepar lobatum" from its lobulated look. The indentations, which are large enough to contain a bean, but are not large enough to put several fingers in, are characteristic of this type of liver.

Fourth. Biliary cirrhosis, or the so-called primary biliary cirrhosis, sometimes called Hanot's disease.

Fifth. The so-called secondary biliary cirrhosis which is not cirrhosis at all, but the result of obstruction of the common bile-duct. When this happens we have interstitial changes which in time produce a big liver. A better name for this type of liver is a literal translation of the German term "Gallen-Stauung Cirrhose," *bile-stasis cirrhosis.*

Sixth. We have a rare type of cirrhosis known as cirrhosis carcinomatosa, a primary carcinoma of the liver. I have put these in about the order of their frequency. The two rare types are the last one and the primary biliary cirrhosis.

Now, as to the differential diagnosis: An alcoholic cirrhosis develops, as a rule, later in life—this man is only thirty-three. Then, too, it occurs as the result of much alcohol. A man who drinks whisky on an empty stomach and has chronic gastro-intestinal disturbance (gastritis, etc.) is the man who is likely to develop this kind of cirrhosis. The liver may be enlarged in the beginning, but as time goes on it becomes smaller and smaller, and then is known as atrophic cirrhosis. As it grows smaller the patient gets worse instead of better. This type of cirrhosis progresses, and we find the principal changes around the portal vein. This is important to keep in mind, because the next symptoms to develop are those due to portal obstruction, *i. e.*, hemorrhoids, stasis in the stomach (so that the patient complains of chronic dyspepsia), and then ascites. Ascites may be the first symptom to be noticed. Jaundice rarely ever develops, is usually a terminal symptom. The conjunctivæ may be slightly tinged, but usually not until the very last stages of the disease.

You can easily see that this is not that kind of a case. The entire course is not that of an alcoholic cirrhosis.

A fatty cirrhosis is not to be considered for similar reasons.

When he first came into the hospital we thought very strongly of a luetic cirrhosis. The Wassermann tests have been negative on the spinal fluid and blood; there is no evidence of syphilis elsewhere; no scars nor other luetic indications. He has had no antiluetic treatment. During the time he has been under observation here we have not observed the slightest symptom that would indicate lues. So we have to abandon the idea of a luetic cirrhosis. There is another reason for its not being a luetic cirrhosis and that is its course. As you see, the jaundice has almost cleared up, and that would not be the case in a luetic cirrhosis if not treated. It generally goes from bad to worse, even under antiluetic treatment, and indeed it is not much influenced by treatment of any sort. There are, however, conspicuous exceptions to this general rule. The smoothness of the liver also speaks against a syphilitic cirrhosis, especially the absence of deep surface indentations.

Next comes the secondary type of biliary cirrhosis. Could this be a secondary biliary cirrhosis due to an obstruction of the bile-duct? No, because if he had an obstruction of the bile-duct sufficient to produce this condition he would be very much worse than he is, and there would probably be other symptoms, such as enlargement of the gall-bladder, etc., which he has not. This patient has had a conspicuous enlargement of the spleen from the very beginning. Furthermore, while the jaundice was at its height the stools were not acholic. The jaundice has now nearly disappeared, but the liver and spleen remain as before.

This bring us down to two conditions—cirrhosis carcinomatosa and Hanot's disease. Now, a cirrhosis carcinomatosa is an ordinary alcoholic cirrhosis with a carcinoma developing on top of it, and that can be ruled out from the course alone. The patient is subjectively improving; the tenderness in the abdomen is much less pronounced, and he is almost ready to go home. He only stayed over because I wanted to present him to you today on account of the rarity of his trouble.

That leaves us Hanot's cirrhosis, sometimes known as Charcot-Hanot's disease. One finds in the text-books very widely varying descriptions of this disease. One author insists that you find a greatly enlarged spleen with a very large liver; another thinks that the spleen is very little enlarged; one author finds that there is much jaundice and another that there is very little. This shows that there are different conceptions of this disease and perhaps a good deal of looseness in its diagnosis. *Genuine Hanot's hypertrophic cirrhosis, in my experience, is really a very rare disease.* I have watched for cases of hypertrophic cirrhosis for the last fifteen years, in fact, ever since I have been in practice; I have gone out of my way to look for them, and I know I have not seen to exceed ten or a dozen cases. I have seen many cases which were supposed to be hypertrophic, but which turned out to be ordinary alcoholic or luetic cirrhoses. A true hypertrophic cirrhosis is best described in the words which Hanot used, *a biliary hypertrophic cirrhosis without ascites and with an icterus.* The characteristic thing about biliary hypertrophic cirrhosis is a very large liver, a smooth liver which does not present great indentations, which is not markedly granular or hob-nailed, and associated with this a big spleen. I have never seen a case which did not have an enlarged spleen—a spleen generally a hand's breath below the costal arch. Coupled with this we find jaundice, which is a striking feature of biliary cirrhosis. The jaundice varies greatly; at times it will be as trifling as in this patient, and again it will be very deep, sometimes almost an olive color. I have seen 2 cases which came to autopsy where the patient's color was almost a light olive green. Jaundice will almost completely disappear and return in a few weeks and be very deep. When this occurs it is characteristic of Hanot's disease.

Life is not materially shortened. I have known several cases which lasted over a decade, whereas other types of cirrhosis, as a rule, run a pretty rapid course, two or three years being usually the limit of life after the disease is discovered. Most of the patients I have seen had a symptom which this patient does not have, and that is an irregular temperature. A

number of them developed a temperature that went up and down a good deal like malarial fever, perhaps reaching as high as 102° or 103° F., and then coming down, and with this temperature there is an exacerbation of the jaundice. This patient has not had the fever which is characteristic of some cases, but in every other way it is a typical case. The liver is enlarged symmetrically; coupled with this is a large, hard spleen; the jaundice came on rather suddenly and tapered off in a few weeks; the patient has remained in good nutrition; all of these things make the diagnosis of a true biliary hypertrophic cirrhosis fairly certain. The aching and pain in the liver are also characteristic of Hanot's disease.

I think we are safe in saying that Hanot's cirrhosis is diagnosticated at least ten times for every time it really exists. I have seen scores of cases which have been supposed to be this disease, but they almost invariably turned out to be an ordinary alcoholic cirrhosis or else a luétic type.

Now what constitute the *salient features in a hypertrophic cirrhosis?* In the first place, it develops in young individuals, has nothing to do with alcohol or lues, nothing certain is known about its etiology, and it has nothing to do with obstruction of the common bile-duct. The principal objective features are a great big, smooth, hard liver with a big, smooth, hard spleen; an onset with jaundice which varies greatly in intensity from time to time, and in some cases an irregular temperature, together with an absence of ascites. These represent the points on which we based the diagnosis in this case. It is a perfectly typical case, except that the temperature has not been as high as is ordinarily the rule. Some authors do not consider the fever as characteristic. It is impossible to say now whether the course is going to be long or short. In spite of the fact that he has been in the hospital for three months his nutrition is good, and we have every reason to suppose that the disease will continue for a long time.

A TUMOR OF THE LEFT KIDNEY—SUBSEQUENTLY OPERATED UPON. OPERATIVE FINDINGS

January 19, 1916.

THE patient I wish to show you is a very typical case in most ways. He is a Norwegian laborer, forty-eight years of age, who came in on the 18th. He complains of passing blood at times in the urine, pain, weakness, loss of weight, and of swelling in the abdomen. He says the trouble began nine months ago, very suddenly he thinks. He passed a large amount of bloody urine, associated with pain and burning after urination. This lasted for two or three weeks, gradually diminishing in amount. He felt well until about four months ago, when he had a second attack; he had a third attack three months ago, and a fourth attack just before Christmas. Between these attacks he noticed no blood. He says he has lost 10 or 15 pounds in weight during the nine months. He never knew that he had a mass in the left side until he was examined here in the hospital yesterday. He had a great amount of trouble in urinating during the last attack.

Past History.—He had a right-sided pleurisy with effusion nine years ago.

Family History.—Father, brother, and two sisters are living and well; mother dead, cause unknown.

The patient has been rather constipated and has taken cathartics freely. There has been no pain or burning on urination except as stated, but he has to get up three or four times at night to urinate. He has been a moderate drinker, smokes a pipe, and uses snuff. He had gonorrhea many years ago; denies luetic infection. He has three children living and well.

Physical examination shows a poorly nourished man who does not seem acutely ill. Regional examination shows that the head is negative, the pupils a little unequal, but they both react to light and accommodation. The neck is negative, chest sym-

metric; the heart shows nothing abnormal, the pulse is about 100, regular; blood-pressure 130 systolic, 80 diastolic. The lungs are absolutely negative.

When we come to the abdomen we see that the left side is distended by a very large mass which reaches away up under the costal arch; below it reaches down below the navel, in front to the median line, and behind goes apparently to the vertebral

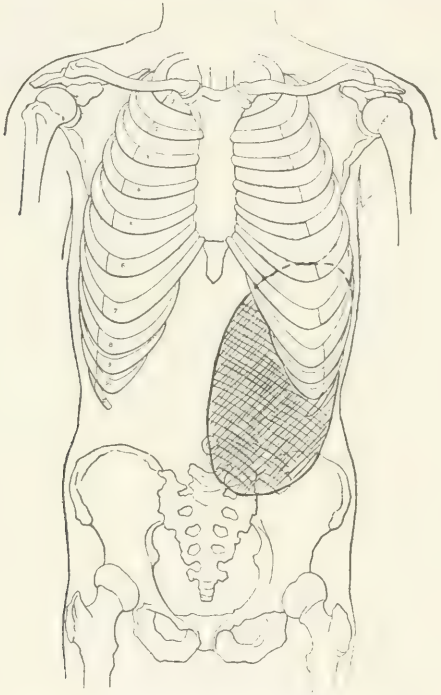

Fig. 218.—Outline of tumor of left kidney.

column. It is hard, quite smooth, very slightly tender, and moves with respiration. No notches can be palpated; there is dulness over the entire mass. The prostate is moderately enlarged. The reflexes and extremities show nothing abnormal. The temperature has been practically normal; indeed, we may say absolutely normal—the course has been afebrile all the way through.

Repeated *urinalyses* show a clear urine, specific gravity varying from 1008 to 1022, no albumin in most of the specimens, occasionally a small quantity, no sugar, no casts. On the other hand, practically every specimen shows twelve or fifteen red cells to a field of a No. 6 Leitz objective; thus, *a few blood-cells are constantly present.*

Blood examination shows 12,200 whites, 4,200,000 reds, 58 per cent. hemoglobin. A differential count shows 61 per cent. polymorphonuclears, 12 per cent. large mononuclears, 2 per cent. eosinophils, the rest small mononuclears.

Now let us consider this tumor a little more closely. In the first place, the abdomen is, as a whole, rather flaccid. In the upper left-hand quarter we see this large mass. As we palpate it it seems to be a single mass. It has some large irregularities on the surface, but no distinct knobs. The edge is rounded, but no notches can be felt.

The first question we must ask ourselves is to what organ does this mass belong? The two organs appearing most likely are, first, the spleen and, second, the kidney. If it is the spleen, it is the size of a very large, leukemic spleen. On the other hand, the blood-picture shows that there is no leukemia. The mass seems to be immediately under the abdominal wall, and as we trace it around it seems to be also *immediately under the flank; it bulges on the side and also in the back.* That makes us think that it is probably not a spleen, because a spleen is usually longer and narrower, and rarely pushes back into the flank as this does. A spleen as large as this would generally come down lower, not so far toward the median line or so far into the flank.

Then I want to call your attention to the form of examination known as *renal ballottement.* If you put your fingers back in the flank and the fingers of the other hand over the tumor, you can get very beautifully this feeling of ballottement with the tumor between your hands (illustrating). This is not absolutely conclusive proof of a renal tumor, but it is very strong evidence at least. The great width and thickness speak in favor of its being a tumor of the kidney. The kidney is, of course, retroperitoneal. The colon passes in front of it, but would be displaced by a large

tumor mass, and this is particularly true of tumors on the left side. If this tumor were of small or moderate size there would be room for the colon in front of it, but an enormous tumor of this size displaces the colon so that it is not only in front of the tumor, but below it. In these large kidney tumors it is much more likely that the colon will be found running right across the lower portion of the tumor or to its inner side. This is not true of a splenic tumor, which generally *pushes the colon behind it.* In this case we have inflated the colon, and it lies right across the lower quarter of the tumor and along its mesial surface. It is, therefore, as nearly as we can judge, a retrocolonic tumor. I think we can regard it quite definitely as being a tumor of the kidney.

There is another point which is important in the differentiation of kidney from splenic enlargements. In enlarged spleens there is generally a band of resonance between the tumor and the vertebral column; *this is not the case with enlargements of the kidney.* In this case (demonstrating) no such resonant area is to be found.

Now, what else can we do to determine if it is a tumor of the kidney? The *x*-ray picture in these cases is usually very satisfactory. The best way to get a good picture is to have some one inject the ureters and take a picture in that way. If we found then on the left side a very large calyx, we would infer that it was a tumor of the kidney. This was done, but the plate met with an accident and we can't show it to you. Dr. Fillis cystoscoped the patient, and found normal, clear urine coming from the right ureter, and from the left ureter a few spurts of blood-tinged urine. Then he injected some indigocarmin, and in a few minutes the right ureter showed the colored urine coming out, but no indigocarmin came through the left kidney for a long time. So we have, then, pretty definite information that the left kidney is diseased. That adds, of course, immeasurably to the probability that this is a kidney tumor—a probability bordering on certainty.

The next question is, What type of tumor is this? The word "tumor" is used here in a broad sense, meaning enlargement. We should first consider the congenital cystic kidney; second, hydronephrosis; third, tuberculosis; fourth, a pyonephrosis of

the left side; fifth, some of the benign tumors of the kidney; sixth, one of the malignant tumors, of which there are several varieties known to occur in the kidney.

Congenital Cystic Kidney.—This mass has a rather peculiar feeling, but we do not find anything corresponding to individual cysts. Then, too, a congenital cystic kidney does not grow with the rapidity with which this has grown. On the other hand, congenital cystic kidneys can have hemorrhages, but the most important thing of all in diagnosing true congenital cystic kidneys is that they are practically always bilateral, and this patient has nothing on the other side. Furthermore, the overwhelming majority of cystic kidneys that are congenital and at all advanced show the picture of a chronic interstitial nephritis. In those cases there is a large amount of urine of a low specific gravity, generally a small amount of albumin, with, of course, a high blood-pressure, and this man has nothing of that sort.

Hydronephrosis.—This may result in a swelling of the kidney and the development of a large cystic tumor. A hydronephrosis may attain a very large size, but, as a rule, some reason will be found for a closing off of the ureter. We know that this ureter is not plugged, for we find bloody urine coming from it in the catheterization of the ureters. Then, hydronephrosis does not grow rapidily, and usually causes more pain. Further, there is not likely to be any blood present in the urine. For these reasons I exclude hydronephrosis.

Tubercular Kidney.—Let us consider this as a possibility. I have never seen a tubercular pyonephrosis anywhere near this size; it is usually two or three times the size of a normal kidney, but nothing like the size of this. Furthermore, the patient has no evidence of tuberculosis elsewhere, either in the genito-urinary organs or in the lungs themselves, *and there is no pus in the urine* —a few red cells were found and that is all. These findings exclude this disease. The same remarks apply to pyonephrosis. The absence of fever is a further point against both tubercular and simple pyonephrosis.

Benign Tumors of the Kidney.—This brings us to a consideration of the benign tumors of the kidney. Practically the

only benign tumor is a lipoma, and this is very rare. I have personally never seen one. It is not likely to grow as this has done; is not likely to cause the loss of 10 or 15 pounds in weight. We do not expect a tumor of the benign type to produce the group of symptoms we have here. In my judgment, therefore, we can rule out a benign tumor of the kidney with perfect certainty.

Malignant Tumors.—That leaves us only the malignant tumors. This mass feels like a sharply outlined, solid tumor. Now, while the man really did not know that he had a tumor until he came into the hospital and his attention was called to it, he knew that his abdomen was enlarging, and he is pretty sure that the whole process does not date back further than nine months when he first had the hemorrhage. Then right from the beginning there was a tendency to hemorrhage—his very first symptom was the hemorrhage. Then the bleeding grew less and less, but did not quite disappear, as he supposed, because it is going on now in the form of microscopic bleeding, of which he knows nothing. So we have the rapid growth and the hemorrhage in favor of malignant tumor. The patient thinks he has lost 16 pounds in weight; this is also indicative of a malignant tumor.

Now, what malignant tumors occur in the kidney? In the first place, there is a congenital sarcoma of the kidney, which we see especially in childhood and which grows very rapidly. That is almost exclusively confined to childhood. Secondly, we have true carcinomata of the kidney. The true carcinomata are extremely rare in the kidney and do not usually attain such a size as this. Lastly, we have the hypernephroma; this, in my experience and judgment, is the typical tumor of the kidney, although some authors question it. It corresponds in type to the suprarenal capsule. Small islands of suprarenal capsule are found in the kidney; these undergo degeneration and develop into a sarcoma-like tumor, which gives a very characteristic picture. It grows very rapidly, it tends to break down very easily, and has a tendency to bleed early and constantly. Therefore, when you are in doubt as to what sort of a tumor is present, repeated

or constant hemorrhages speak rather in favor of a hypernephroma than otherwise.

There is not much to be gained from a clinical standpoint by discussing the pathology of these malignant tumors. My own feeling is that the great majority of malignant kidney tumors seen in adult life are hypernephromata. The histologic details of the tumor have very little clinical interest for us. In my judgment this patient has a malignant tumor of the kidney, a sarcoma-like neoplasm, and, probably, that variety known as hypernephroma.

These cases are not so very rare, but one does not often see them of such size as this without having attracted more attention on the part of the patient. Let me call your attention to two or three things in regard to these tumors. *Hypernephromata become generalized largely through the blood-vessels*, and we may find metastases not in the liver, where we might expect to find them, but in the lungs. I have seen several cases where the metastases were practically confined to the lungs. So the thing to do in every such case is to examine the lungs very carefully. In this patient we can find absolutely nothing abnormal in the lungs, and there is no enlargement of the liver or glands. We think, therefore, that there is some chance of completely removing the tumor by operative interference.

Pain in these tumors is not as invariable as one might think. There may be traction pain from dragging of the tumor upon the nerves or there may even be colic at times. Blood accumulates in the pelvis of the kidney and long clots are forced down the ureters, and the patient has attacks which are very much like a stone colic. Then it is possible that small necrotic pieces of the tumor itself may get into the ureter, but that is very rare.

As to the course and termination of a case like this, the tumor usually grows rather rapidly, and if not removed, metastases form, generally in the lungs, and the patient succumbs to the general metastases. If the tumor is confined entirely to the kidney the chances for recovery after nephrectomy are fair. I have not been able to make out any metastases in this case, but I should not be at all surprised if there were many small metastases pres-

ent. It is impossible to locate small metastases in the lungs or in the glands, but the sooner he is operated the better it will be for him. We know that in many cases where we cannot, clinically, find any metastases, they are found on operation in various places where they have eluded observation.

I brought this man in because Dr. Andrews is going to operate on him at the close of my clinic. Those of you who are interested will have the opportunity of seeing whether the diagnosis in this case rests on a sound basis or not. (Dismisses the patient.) Now that the man is out of the room, I will say that I think it is very doubtful whether they will be able to remove the tumor. There are probably metastases which we cannot demonstrate clinically. A tumor of this size probably has metastases somewhere, but operation offers the only hope for the patient.

NOTE.—On operation the tumor was found to be a malignant neoplasm of the kidney. There were one or two large areas of necrosis in it, softened areas filled with blood, and necrotic masses. A large number of the glands in the surrounding region were enlarged. Dr. Andrews decided not to attempt the removal of the tumor. So far as could be judged, it was a hypernephroma.

AN ABDOMINAL TUMOR IN THE POSITION OF THE RIGHT KIDNEY AND RETROPERITONEAL, BUT NOT CONNECTED WITH THE KIDNEY

THIS patient is a Bulgarian, twenty-nine years of age, a laborer, who came into the hospital on the 29th of December because of pain in the lumbar region and considerable loss of

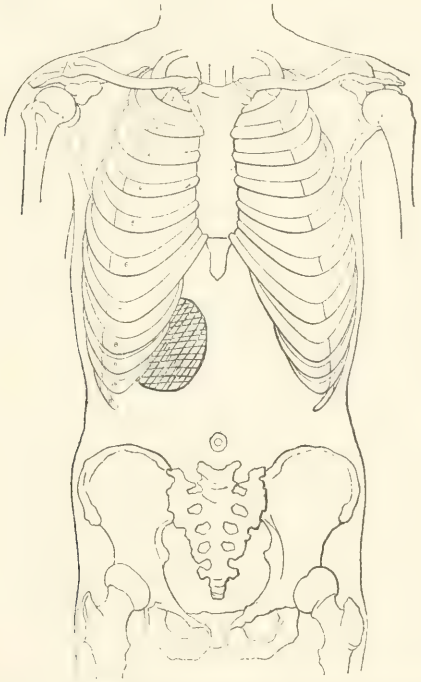

Fig. 219.—Outline of retroperitoneal tumor not connected with kidney.

weight. He has been sick for three months. The pain in the right lumbar region has been severe ever since the onset and has recently been in the left lumbar region also. He has lost about 23 pounds in the last three months. He has to get up several

times at night to urinate, but has no burning sensation on urination. He has not worked since last September on account of the pain.

His father, mother, four sisters, and four brothers are living and well. He is married and has three children, all living and well. Venereal infections are denied.

Physical examination shows that he is fairly well nourished, but very markedly bronzed. I am not sufficiently familiar with the natural color of the people of this race to know whether he is abnormally bronzed, but certainly some areas on the abdomen look very much pigmented. The head is negative; the neck shows a few little glands. Blood-pressure is 160 systolic, 80 diastolic. The liver and spleen cannot be palpated. When we go deep down in the right side of the abdomen we find a mass. As you inspect the abdomen you will see that it is sunken in and retracted. In the right hypochondriac region there is a good deal of tenderness most of the time and we feel a very interesting tumor. It seems hard and reaches down to about an inch above the navel, extending an inch or so from under the ribs. It moves very little, if at all, and seems to be close to the vertebral column; it gives the impression of being almost fused with the vertebral column and is very hard. To the outside of this mass laterally one can feel something which is possibly the kidney pushed out to the right. I wouldn't dare make such a statement if we didn't have an *x*-ray which suggested this possibility to us. This patient's ureters have been catheterized and a silver salt injected. (Demonstrating *x*-ray.) You can see, on the right side, the ureter going upward and outward, and the *shadow of the pelvis being a good deal further out than it is on the left.* Probably what we feel to the side of the mass is the kidney pushed away out to the right by the tumor mass.

Our first impression was that the tumor might be the kidney, but when we had the urinalyses made they did not show what we expect to find in a kidney tumor. The urine was amber colored, contained a merely perceptible trace of albumin, only an occasional leukocyte, and an occasional hyaline cast, nothing especially abnormal. At the time the ureters were injected the

urine from the right kidney showed a mere trace of albumin and that from the left showed no albumin, but a good many red

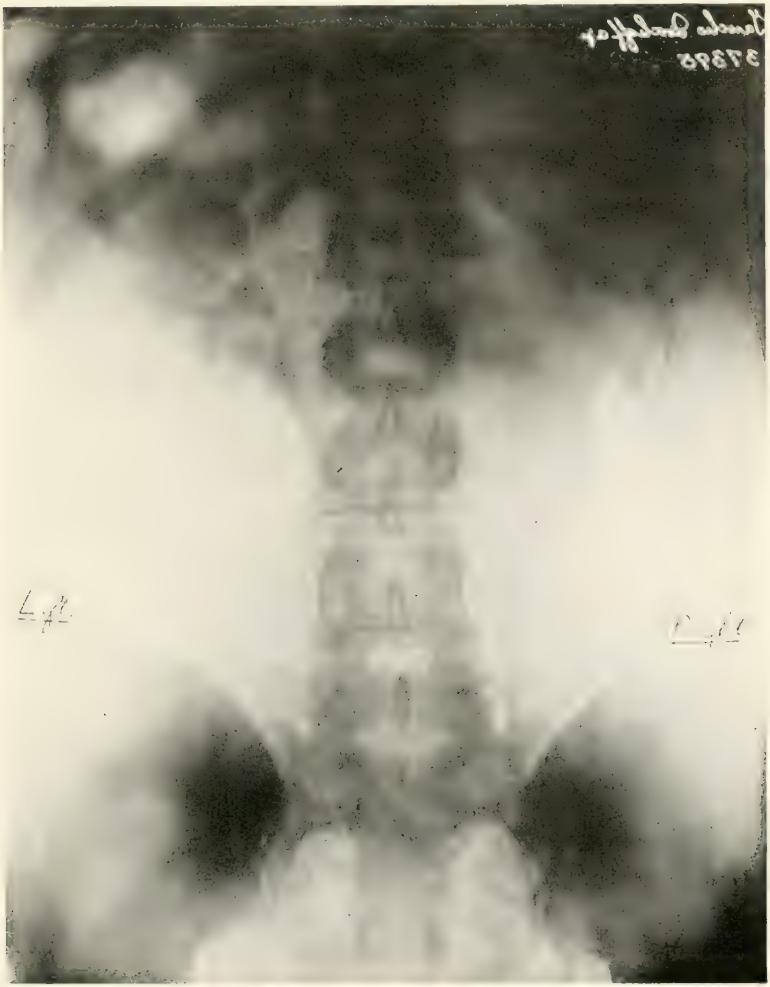

Fig. 220.—Kidneys injected with silver solution, showing right kidney pushed outward by tumor.

cells which were probably due to the trauma. Then the Löffler examination for tubercle bacilli was made on the urine and nothing found. Blood examination showed 11,300 whites, 5,300,000

reds; the differential count showed 81 per cent. polymorphonuclears.

Now, what sort of a diagnosis are we to make in this case? I did not mention that we had examined the stomach contents and stools. We removed 180 c.c. of stomach contents after a test-breakfast; there was no free hydrochloric and 33 per cent. total acidity; also no blood and no Gram-positive bacilli were present. The *x*-ray findings were negative.

There is in this case a very definite tumor mass, apparently retroperitoneal, in the usual position of the kidney, but in this case pushing the kidney out to the right. The tumor is exceedingly hard, seems to be slightly nodular, and does not move with inspiration to any noticeable degree. Apparently it has all the earmarks of a retroperitoneal tumor. What more can we do to ascertain the organ to which this tumor may belong? The first thing I am going to suggest is that we try to inflate the colon and see if it goes in front of the tumor or if the tumor is in front of the colon.

In inflating the colon have the patient well relaxed, inflate it very slowly, knead it in the sigmoid flexure and in the splenic area, and in that way you can get the air to go in better and get the larger part of the colon inflated. This was done yesterday, but was not very successful, and caused him a great deal of pain, but we are going to try it again today. (Intern begins to inflate the colon.) You can use an ordinary catheter slipped into the rectum. The best thing to use for the inflation is one of the double bulb arrangements from a Paquelin cautery. The idea of kneading the bowel as you inflate it is to keep it from kinking at the flexures. Now let us note that the percussion sound over the tumor is definitely dull. The descending colon is inflated, and now, as we inflate the transverse colon, you notice that the note is getting louder and more tympanitic, and the tumor is very difficult to feel because apparently the colon runs in front of it. Thanks to the kneading, we are getting in a great amount of air. There can now be no doubt that the tumor is back of the colon—a retroperitoneal tumor.

As far as we have gone, I think we are justified in saying that

the man has a slowly growing very hard tumor, situated just to the right of and closely fixed to the vertebral column, occupying almost the position of the normal kidney. It pushes the kidney out on account of its size. I think at present we are not justified in going further, but I will try to show him to you at the next clinic hour, and by that time I hope we will be able to make the necessary examinations to find which organ this tumor is connected with. It is just possible that it is the pancreas, although it is a little low. It is also possible that it might be an adrenal tumor.

(Additional note, *January 12th.*)

The first abdominal tumor case I showed you last week is to be operated today. You will remember that I said at that time that the tumor was retroperitoneal, and it seemed pretty certain it was not the kidney, because when we injected the kidney the shadow was pushed further out. Afterward, when we got his bowels more thoroughly emptied, we could feel his kidney far outside the normal line, so that the diagnosis resolved itself into a retroperitoneal tumor, in all probability malignant. Whether it is a primary tumor in this locality we cannot say; we have studied the case for a whole week, but have been able to find nothing new. We have no reason for supposing it to be connected with stomach, pancreas, or gall-bladder. The patient had a negative Wassermann. He was suffering so intensely that life seemed unbearable to him. My surgical colleague has agreed to operate, although I question very much whether any benefit will come from the operation because the tumor is so deep seated. If it is malignant, as I think it undoubtedly is, there are probably metastases elsewhere. If the doctor cannot excise the tumor, I shall try to get him to take out a little piece for microscopic examination.

(Additional note, *January 19th.*)

I imagine most of you did not see the result in this patient who was operated upon last week. No more information could be obtained than we already had. You will remember that there was a hard, nodular tumor situated close to the vertebral col-

umn, which we believed was not in connection with the kidney, the stomach, or any of the abdominal viscera. That proved to be the case. Dr. Andrews made a big incision, and we could see that the tumor had no connection with the pancreas, stomach, or kidney. The kidney was pushed away out to the side. The tumor seemed to take its origin in the retroperitoneal fascia. It was found to be absolutely inoperable, and the doctor did not feel justified in removing a piece for diagnosis. It seemed almost certainly malignant, probably a sarcoma. So our diagnosis as far as it could go was absolutely correct.

CLINIC OF DR. JOSEPH ZEISLER

NORTHWESTERN UNIVERSITY MEDICAL SCHOOL

PRESENTATION OF A CASE OF MYCOSIS FUNGOIDES

With Special Reference to Differential Diagnosis and Treatment.

THE case which I am about to present to you, gentlemen, is one of unusual interest and unusual importance. If, by way of preface, I tell you that this poor young woman has been treated for a considerable period for a supposed eczema, and if you learn later that she is afflicted by one of the most serious, not to say fatal, diseases of the skin, you will recognize the importance of the proper diagnosis in cases which begin with an apparently harmless and insignificant picture.

If I call your attention to the small lesion which this patient has in the left axillary fold, and to another similar spot here on the right elbow bend—harmless looking efflorescences—and if you compare with these the enormous picture which the right axilla presents, and the one which you will presently observe in the right groin, you have at once the two antipodal points of this malady—you have the placid overture and the extremely tragic finale of this symphony of pathologic features.

History.—This young lady is thirty years old, and has had this trouble for about five years. It began as evanescent, roundish disks on the face and soon appeared on the neck, but, as I intimated, these spots disappeared and others formed elsewhere. They always gave rise to such fearful itching and burning that she could not rest at night and had to resort to the use of whisky. More recently the very grotesque condition which you notice in the axilla developed, a condition intensified not merely by pure surface phenomena, but by the formation of distinct tumors. She has always felt perfectly well and she

feels practically well now. There was no disease of any importance which preceded the present malady, nor are any of her vital organs at the present moment affected to any appreciable degree.

Fig. 221.—Case of mycosis fungoides.

I have had her urine examined. After standing for only a short time the previously clear urine assumes a brick-red color. An enormously heavy sediment is deposited, which, upon examination, is found to consist principally of urates. This is proved by the fact that heating dissolves the deposit very readily.

It might also interest you to know something of the *blood-*

picture. A blood-count made a week ago showed 11,400 leukocytes. So far as the differential count is concerned, we find small lymphocytes 30 per cent., large lymphocytes 7 per cent., polymorphonuclears 58 per cent., and eosinophils 5 per cent. These proportions do not indicate anything particularly significant. A slight increase of eosinophils is found in a great many dermatoses and does not mean anything distinctive. The leuko-

Fig. 222.—Case of mycosis fungoides.

cyte count does not correspond to the findings in leukemia or allied conditions—a point which will be appreciated when we speak of the diagnosis and the differentiation of this disease from other similar dermatoses.

Perhaps it may also be worth mentioning that her case has been pronounced carcinomatous by some, tuberculous by others, and has been treated as an aggravated form of eczema. I have shown you the small, roundish, reddish, slightly scaly

spots disseminated in various locations. I have shown you this large area involving the right axilla (Fig. 221), and will now show you for a brief moment the much larger diseased area located in the right inguinal region (Fig. 222). There is a distinct, irregular patch, and besides a large, erythematous, scaly area of peculiarly grotesque, not to say fantastic, shape, which in some places shows definite infiltration. You will notice that some of these patches have a tendency to ooze, and a week ago the patch in the axilla was secreting so profusely that the patient had to change the dressing over it several times a day, but since then, as the result of treatment, it has dried up markedly.

The first question which must interest you is, What have you before you? This is a case of *mycosis fungoides*, also called *granuloma fungoides*. This disease is comparatively rare, although dermatologic literature has recorded quite a considerable number of cases in recent years. The disease was described as early as 1814 by the French dermatologist, Alibert, under the name of "Pian fongoïde," which he later changed to "mycose fongoïde," indicating the tendency of the infiltrated areas to develop these mushroom-like excrescences and flat tumor formations which are so characteristic of this disease. But cases have been described under a variety of names, as "granuloma fungoides, "lymphodermia perniciosa," "sarcomatosis generalis," "eczema tuberosum," etc.

The case before you is particularly instructive, because it illustrates the three stages which have always been recognized in this disease. An experienced dermatologist can often foretell the eventual tumor formation from the appearance of the skin in the premycotic stage. This state is indicated here by the few roundish, slightly scaly patches which I have shown you in various places on the surface. Mycosis fungoides starts in by just such insignificant looking patches, which are easily taken for eczema, for psoriasis, for pityriasis rosea, which it resembles somewhat in these oval or ringworm-like formations, and it reminds one, to a certain extent, of that ill-understood but well-known dermatosis which French dermatologists described under the grandiloquent term "erythrodermie pity-

riasique en plaques disseminees," which is supposed in the title to comprise all the different features of the disease.

Diagnosis.—In the last number (February) of the "Journal of Cutaneous Diseases," which reached me only two days ago, there is a splendid article by Wise and Rosen on the differential diagnosis of this very dermatosis, which is also known as "parapsoriasis," from mycosis fungoides. This article refers particularly to the histologic picture of the two, and shows that, while clinically the diagnosis may be very difficult, microscopically it is easy to keep the two diseases well apart. From what has been said it is quite conceivable that mistakes, in the early stages of this disease, are often unavoidable. It takes almost a prophetic look into the future for the experienced dermatologist to recognize readily in this premycotic stage the future development. These flat patches, then, are the first stage. The second stage comprises the marked infiltrations, as you find well illustrated in various places here. Anywhere in this axilla you may note the infiltration which precedes the eventual tumor formation, for instance, right on the borders of this patch (demonstrating). From this second stage, which microscopically is characterized by unmistakable features, there is only one brief step to the third stage, the tumor formation, of which our case presents several examples—one in the right groin, a flat but distinctly raised tumor, and smaller, more sharply circumscribed tumors in the right axilla, which show a distinct tendency to ulceration.

Fortunately for this young lady this is a comparatively mild case. If you will look at the picture which Stelwagon gives in his text-book of one of his own cases, and see there the dozens, even hundreds, of tumors covering the face, the extremities, and the body, tumors which spring up from the skin mushroom-like, and with a distinct tendency to ulceration and decay, you will form a fair idea of the severe cases. Another good illustration you may find in Ormsby's text-book: a patient literally covered with tumors, in whom the whole hand had to be amputated. In this young lady most of the surface of the legs and the arms, the chest and back is free from tumors, showing that her case is not as yet in that catagory, but it is very characteristic, and for

the beginner as typical an example as could be given, because it shows distinctly the primary patches, the infiltration stage, and the tumor development.

Prognosis.—I hope this patient will not take it too much to heart if I mention the usual fate of this disease. If left alone, if untreated, these cases have a sad outlook. The expectancy is that more and more tumors will form, even without the preceding flat patches, that life may be much shortened, probably by secondary processes, which is natural enough with any numerous tumor formation in which there is a tendency to disintegration through the absorption of septic material. Still, even severe cases have been known to live as long as fifteen years. In recent times the prognosis for these cases has been very much improved through the treatment which we have learned to employ.

A question of great interest is the **nature of these tumors.** What are they? As careful an observer and accomplished clinician as Kaposi considered them as sarcomas, and such cases were often described as generalized sarcomatosis. True, the histologic picture shows a great resemblance to the histology of the round-celled sarcoma. Others have been inclined to look upon these tumors as lymph tumors, as lymphodermata, and they were strengthened in their opinion by the peculiar blood-picture which occasionally resembles that found in leukemia very strongly. The overwhelming opinion of observers is, however, that we have to do with a peculiar form of granuloma, a view which was first pronounced in authoritative manner by Hochsinger and Schiff in a splendid monograph, published in 1886 in the "Archives of Dermatology and Syphilis," and which has been corroborated by many others. There are some authors who look upon these tumors as neither of sarcomatous or lymphomatous or granulomatous character, but as peculiar idiopathic tumors which are found only in this disease.

The fact that these tumors sometimes disappear spontaneously speaks strongly against their sarcomatous nature. Their ready response to therapeutic measures also stamps them as something less serious than we are accustomed to look upon in sarcoma.

The diagnosis in a case of the sort you see before you is really child's play, I am frank to say. When I saw this patient for the first time a week ago the diagnosis came to my mind as quick as a flash. A younger colleague, to whom I showed the case at the time, hesitated and was not quite sure. This case, as I say, is easily diagnosed, because it is the most typical case you could possibly imagine, and yet there are instances where the differentiation from other diseases is not so easy, particularly in the premycotic stage. But let me remind you of the one important feature during this stage, the intense pruritus. I might say the unreasonable amount of itching which these patches cause should at once give rise to suspicion. No case of eczema ever itches so much or ever produces such peculiar, gyrate, characteristic shapes as you see here. The resemblance to the other dermatoses mentioned before is a matter of great importance and of frequent occurrence. When we read the history of many cases which during the early periods were utterly misunderstood we can realize the difficulties of the early diagnosis. How one can think of carcinoma in such a case it is difficult to see. Carcinoma does not produce such large, flat areas; the massive infiltrating forms of carcinoma do not produce any protuberant lesions upon the surface of the skin itself, and other forms of cutaneous carcinoma are characterized by the hard pearly border which is utterly absent here.

Tuberculosis of the skin is known to you in many of its forms, and, while it may result in ulceration, may produce nodules, it never causes these sessile, pedunculated tumors as you see them here. If we had a picture here of generalized brownish patches one might think of leprosy, in which we know there is a tendency to the formation of small tumors with subsequent ulceration. But there is nothing present here to substantiate such a notion. I am not able to present a histologic picture of this case, because I did not consider it necessary for the diagnosis and did not care to subject this private patient to inconvenience.

Treatment.—When we consider the almost inevitable fatality of this disease in former years, and compare with it the great improvement which it is possible to achieve at the present

time, we may appreciate the great accomplishments of modern dermatologic therapeutics. The supreme remedy on which we rely in combating this disease is the Röntgen ray. Few and mild exposures very often cause a complete involution not only of the infiltrated patches, but a gradual shrinkage and finally a total disappearance of the tumors. The four treatments which this young woman has had within the last week have, even in this short time, produced a remarkable change, having caused a drying up of the intensely oozing surface and having brought about a definite flattening of the protruding places. She is going to be given the benefit of this treatment not only for the tumors, but for the smaller patches, and I may be able to bring her to you again and show you the result of the treatment. We expect great improvement.

In addition to these radiations we rely upon the use of arsenic, either internally or hypodermically. Arsenic, started with small doses and increased to a point of physiologic tolerance, maintained at this point and then gradually reduced, has given good results in some cases even in the ante-Röntgen-ray era. The thought naturally has occurred to some observers that arsenic, in the form of salvarsan or neosalvarsan, introduced intravenously should have a striking effect. It has been given a trial repeatedly and seems occasionally to have had a temporary effect, but nothing brilliant has been reported so far. The internal use of arsenic, as you know, often has a disagreeable effect upon the intestinal tract, besides general toxic phenomena. It is, therefore, preferable to use hypodermic injections, particularly of cacodylate of soda, which can quickly be pushed from 1-grain doses up to 5-grain doses or even more. This is also being tried in the case of this young lady, and I hope that the combination of the arsenic treatment with the Röntgen ray will accomplish much for her.

Note.—As a result of the treatment there has been a remarkable improvement up to the present date, April 1st. The tumors and infiltrated areas have almost completely disappeared.

CLINIC OF DR. RALPH C. HAMILL

NORTHWESTERN UNIVERSITY MEDICAL SCHOOL AND WESLEY HOSPITAL

TRAUMATIC NEUROSIS

Analysis of Its Mechanism and Development Based on the Etiologic and Clinical Factors. Consideration of the Mental and Physical Processes Involved.

Thorough Understanding of the "Attacks of Apprehension" Necessary to an Intelligent Insight Into the Disease. Early and Late Symptoms of the Phenomenon and its Association with Phobias.

Marked Susceptibility to Suggestion and its Recognition of Importance in the Diagnosis.

Fundamental Factors in the Development of the Neurotic Individual.

Psychology of the Adolescent.

Treatment of These Cases.

February 28, 1916.

ONE of the subjects in clinical neurology that is as difficult to comprehend as any is that of traumatic neurosis. The importance of the subject is appreciated when its close relationship to hysteria, neurasthenia, and all functional nervous conditions is realized. Today I am going to show you a patient who is suffering from a traumatic neurosis; vomiting is her principal complaint at present.

Traumatic neurosis is a functional condition for which sufficient organic basis cannot be discovered.

The principal complaints registered in a traumatic neurosis are divisible into two classes—general and local.

The general are depression, nervousness or restlessness, poor sleep, easy fatigue, failure of concentration and poor memory, irritability, headaches, dizziness or lightheadedness—such complaints as these, all of them spoken of as being almost unbearable, and yet described in a manner which you realize is somewhat intensified by the individual's cognizance of his own symptoms; that is, in the telling it is apparent that the patient is emphasizing his trouble as strongly as possible.

The most important of the general manifestations in a traumatic neurosis are the attacks of apprehension. A thorough understanding of these attacks is necessary if one wishes to gain an intelligent insight into the symptomatology of the traumatic neuroses. They also form a broad connecting link between these exogenous neuroses and neurasthenia and hysteria.

First of all, it must be realized that apprehension is fear; fear as to what may happen. These attacks, then, are evidences of fear. They consist of the physical manifestations of fear; their symptoms are the reactions of the body to a state of mind.

It is quite possible that this state of mind has a physical cause—a chronic intoxication, overfatigue, developmental defect, etc. Be that as it may, in our endeavor to work toward the origin of these neuroses we must first of all understand the dependence of the complaints of the patient on their sources, *i. e.*, on the mental state of fear. A recital of the symptoms of an apprehensive attack is merely to speak of the various ways in which fear affects the body.

These attacks of apprehension in their entirety, as they seldom are except at the beginning of the trouble, are marked by a feeling of palpitation, a throbbing of the heart, a sense of smothering, a choking sensation, some weight on the chest, a feeling of light-headedness or of heat in the head, cold feet and hands, a sinking sensation in the abdomen, perhaps the cold sweat breaking out all over the body, a tremor, a weakness behind the knees and in the legs, a sense of everything giving way—in other words, the symptoms of sudden, overpowering fear. The various symptoms that are found in these attacks of apprehension may all be present, and usually are early in the condition, whereas later there

may be only one or two of them to mark the onset of such an attack.

All these complaints are easily recognized as signs of fear by anyone not suffering them, but to the patient they represent something wrong with the part of the body in which they are felt. They are translated by him into signs of organic disease. Upon the recognition of this translation depends the first step toward an understanding of the neurosis. This is true for the patient as well as for the physician. Patients will say that they have heart disease because they have attacks of palpitation; or a goiter, an aneurysm, or a stricture in the throat because they can't breathe and have smothering attacks; or they will say that they have become weak because of the feeling in the knees; or that they have poor circulation because their feet are cold; or they claim they have indigestion because of the sinking sensation in the stomach.

I can't overemphasize these attacks of apprehension; I don't believe it is possible to overestimate them in medicine. There are more of these people being treated under mistaken diagnoses than any other disease known in medicine. Operations, stomach washings, prescriptions of tonics, tincture of digitalis, etc., without number are being used in the treatment of neuroses. I cannot say that they are badly used in all cases. If the patient has sufficient faith in the physician and the physician seems to know what he's about, the patient oftentimes feels well cared for and the natural interest in life, reasserting itself, finds a responsiveness impossible in the apprehensive individual for whom nothing is being done.

These apprehensive attacks are associated with other fears which we speak of as *phobias;* that is, a person has a phobia about a crowd—he goes into a crowd, has smothering sensations or possibly gets nauseated, and has to get out of it; such crowds are those of the theater, church, or the street. And these patients avoid them. Another phobia is the phobia of open spaces: these people will be very much afraid to cross the street; so long as there are others close around them they will be all right, but the moment the space opens up a little they become terrified and

afraid they will fall. The fear of elevated trains or going up in elevators in high buildings is another phobia. Another one has to do with any method of transportation, such as the street car, railway train, or elevated train. The fear of being left alone is another phobia that is very common; these people will not allow themselves to be left alone, but will make a fuss so as to keep somebody constantly by them. These are the principal mental aspects of the traumatic neurotic.

Perhaps I have not spoken enough of the restlessness and sleeplessness, especially the latter, of which they complain so bitterly, and which are associated with the attacks of apprehension. The sufferer gets into bed, the lights are turned out and there is nothing about to distract him; so he lies awake thinking of himself, gets more and more wrought up, and then he gets scared and calls for help. Or, if the process does not go that far, the fear that he is not going to sleep becomes just as much of a phobia as the attacks of heart disease, and because he thinks he is not going to sleep and is so afraid he will not sleep, he does not go to sleep; or he will wake up frequently during the night. While he may sleep enough, so far as the total time is concerned, he wakes so frequently and the periods of wakefulness are so marked that he feels as though he had not slept at all. You will frequently see a person, looking hale and hearty, with no black circles about the eyes or anything to indicate loss of sleep, who will complain that he has not slept for a month, when it is perfectly apparent that he has had plenty of sleep. These same people will complain of restlessness; they can't do one thing long at a time, can't sit down and read a book, and because of the depression which comes over them they suffer intensely. The depression perhaps is not so marked in the traumatic neuroses as in some of the other functional conditions, but it still is of considerable importance. Irritability is also a prominent symptom.

Their ready fatigue is of a great deal of importance in the sense that because they have no organic defect their physicians and friends and family tell them there is nothing the matter with them and that they should go back to work. They return to work and find that they can't work; they tire out, and because they tire so

easily they overemphasize their symptoms, for the reason that their symptoms form the only excuse they have for not working. The employer will not accept the excuse "I'm tired" for failure to work for which they are paid. So these individuals make their excuses in terms of symptoms of disease—they have heart trouble or indigestion or a goiter—rather than in terms of merely being fatigued.

Local Symptoms.—Among the symptoms of traumatic neurosis that might be called local as against these general ones is the loss of function or paralysis of a hand or leg or foot; loss of sensation, an anesthesia, or hypesthesia. The patient that I am going to show you has a left-sided hemihypesthesia. A very characteristic thing is that they will say they were struck on one side of the body and that the spot has always been tender. You can emphasize to them the tenderness, say, on the left side of the abdomen, and if you stick them with a pin on the right side they will say it is sharp, but when you stick them on the left side they will say it is dull or they do not feel it; that is, you can suggest to them that because they have been hurt on the left side but were uninjured on the right side, you expect them to feel differently on the left side, and as a consequence they have a hypesthesia. Along with that loss of superficial sensation they will say that when you press deeply on the side that was injured it hurts more than on the other side; that is a very characteristic finding. This combination of superficial anesthesia with deep hyperesthesia is similar to the sensory disturbance seen in an alcoholic neuritis.

Along with this there may be loss of the sense of sight or hearing or taste or an aphonia (a loss of speech). The loss of the power of speech is one of the things that commonly results from a bad accident.

The pains that these patients complain of are very frequently in the *lower part of the back*, which is quite natural, because their injuries are very likely to occur in such a way as to give them a shock which they feel along the spine. The small of the back is a place that is very readily wrenched or strained, and it is very frequently complained of, especially in women. Hence, the

"ambulance chaser" is led to tell a woman patient that she has a displacement of the uterus. That was what happened in this patient; before the doctor ever made a vaginal or rectal examination he did a ventral fixation because "she must have some displacement"!

In a man very often *scrotal pains* are complained of, or varicocele, and of course these pains are dwelt upon by the patient, his doctor, and his lawyer. In the male, loss of potency is frequently complained of after an accident. Men who are apprehensive will often complain of impotency, but they base it on the supposition that they had some local injury; their loss of potency is real, but it is because of their apprehension and not on account of the trauma.

I should also have spoken of *coccygodynia* (pain in the coccyx), with inability to sit for any length of time without great discomfort, a frequent complaint in all these neuroses, especially when the patient was sitting at the time of the accident or when he has fallen on the buttocks.

History of Patient.—This patient's history is as follows: She came into Wesley Hospital on the 29th of January complaining of pain in the abdomen after eating and of headache. She said the trouble started three weeks before. She was eating a light meal, when she had a pain in the upper part of the abdomen associated with vomiting. It lasted for five or six hours; she had been constipated before this spell came on. She felt some better the next day, but called a physician. She did not get rid of the trouble and as soon as she would eat vomiting would follow. During the past two weeks the trouble has remained about the same and she has been unable to keep food down. She says the attacks of pain last for ten or fifteen minutes and then pass away. She also complains of tenderness in the epigastrium, of vomiting, and of a frontal headache.

As to past illnesses, she told the intern she had small-pox at three, pneumonia during infancy, and mumps at nine years of age.

It happened that she had been a patient on my service at the Cook County Hospital about eight months ago; she recalled that

fact to me and I looked up her history. Although she says she had her tonsils and adenoids removed the latter part of August at the County Hospital, she tells nothing of the trouble that took her there. She went to the County Hospital because she fainted, fell and hit her head, and following this fainting spell she had delusions that she was seeing a forest fire, that she was traveling, that she was with her "young man," who, as a matter of fact, had gone to the Hawaiian Islands as a soldier some months before.

There is nothing in the history she has given here about an accident, and nothing about the possible relation between the vomiting and the accident, but when you dig into the history a little you elicit the following facts: That she has had apprehensive attacks for the last five years; she is now twenty-two years old. Three of them may have been more than merely apprehensive attacks, because after having the usual symptoms of an apprehensive attack she says she lost consciousness. The attacks started when she was seventeen. She had been at work for a short time when the first one occurred; her menstrual period had just come on. She was very tired when she came home from work; she laid down for a little while, and her mother persuaded her to take a little supper. She had a buzzing in her head and felt dizzy and fainted. Following that she slept poorly and had a headache the next day. Then she had attacks of palpitation off and on for a couple of years, but nothing of importance until two years later, when she was in the tornado at Omaha. The roof was taken off the house. She was menstruating at the time, had one of these bad apprehensive attacks, became more and more frightened, and finally fainted again.

The third bad attack was associated with a street-car accident. That seems to be the start of her present trouble. The accident occurred on Decoration Day, 1914; she had just started to menstruate; there were several cars in a line, and she had just said to a woman who sat beside her that if they didn't look out those cars would bump, when sure enough just then they did bump. She was thrown against the back of the seat in front of her, back against her own seat, toppled off the car, and landed on

her buttocks. She probably had risen partly to her feet and finally "sat" off the car rather than fell off. She struck her head against the stanchion of the car as she fell. She was unconscious for three hours, and was in bed for twenty-nine days afterward. About five days after the accident she began to have pains in the back, running down into the vagina, and the doctor said she had a bad displacement. These pains continued until she was operated upon last August, when a ventral fixation was done.

The fourth bad attack was last summer. The first attack was when she was about seventeen, at the time she first started to work; the second came during the tornado; the third was associated with the street car accident—she had apprehensive feelings when she saw the cars so close together, the accident occurred, and ever since she has had pains, nausea, and a tendency to vomit. Then, associated with the last attack, or to be considered in its etiology, is this fact: When she was convalescing from the operation for uterine displacement she had to leave the hospital before she was entirely well, to go home and help her aunt take care of the aunt's young children. The children, of course, had no consideration for her; they were a constant source of irritation to her. She thought of herself as a sufferer, one who should be cared for, considered, and spared. In truth, she was. She was still weak from the operation. Her physical and mental fatigue were perfectly real and reasonable. Yet in this state of fatigue, instead of being waited on and tenderly considered, she was obliged to meet the whims of and humor someone's else children.

Physically and mentally this was too much for her, so when the break came—when the next apprehensive attack occurred—she gave up altogether, and what was formerly only nausea became vomiting, what was fatigue became prostration.

Reflexes.—One of the things constantly seen in these apprehensive conditions is the brisk reflexes. You can all see how very brisk they are in this case (demonstrating); there is even a slight tendency to a foot-clonus; it stops very suddenly, is very unequal, and is not demonstrable every time by any manner of means.

The same is true of the knee reflex; there are a few jerks, such as we see in the foot-clonus. If this were a true ankle-clonus, an organic affair, we would probably have a Babinski reflex, but, as you can see, there is a perfectly normal plantar reflex here.

You can also see that there is a marked difference in the effect of a pin prick on the two sides. It is usually recognized as sharp on either side, but whereas it is borne without complaint or distress on the right side, it is winced from and feared on the left. Along with this difference in superficial sensation there is an immediate complaint of pain on squeezing the tendo achillis, but she says it hurts a little worse on the left side. There is no particular difference to touch on the two sides, but there is to pain. As a matter of fact, I think I suggested this hypesthesia at the County Hospital nine months ago. It was a question then of making a diagnosis, and I found this hemi-anesthesia and brisk reflexes. They are associated with no other changes. One might think of multiple sclerosis, but the abdominal reflexes are normal, the reflexes in the arms are not particularly exaggerated, and the rest of the neurologic examination is negative.

I say "it was a question of making a diagnosis, and so I proved this hemi-anesthesia" for this reason. The finding of the sensory disturbance was entirely a matter of being able to suggest its presence. The patient realized I expected to find a change in sensation and so responded to my expectation. She proved thereby to have a considerably increased suggestibility. It is this suggestibility that is of diagnostic importance. Suggestibility in this instance means that because of an idea or a state of mind a physical or bodily symptom comes into existence. It is just this causal relationship between states of mind and physical symptoms that forms the foundation upon which all the symptoms of the neuroses are constructed. Hence the demonstration of suggestibility is of diagnostic importance.

This young woman has had her stomach washed out I do not know how many times—so many that, like an old shirt, it ought to be worn out by now. She has had test-meals repeatedly and

they have been perfectly normal. All the other examinations have been negative.

I was sitting by her bedside one day when her meal was brought to her. She drank a little soup and then obviously swallowed air. She was talking pleasantly to me at the time (I mention this to show that she was in no distress and suffering no discomfort), but she was swallowing air, and after she did that five or six times she reached over for the pan and vomited what she had eaten. She does not look toxic, or emaciated, or very ill, and yet she has been vomiting for the past seven weeks. But, as I say, when you watch her eating you see that she swallows air, and then pretty soon and very easily out comes some of the food, and that constitutes her bad vomiting.

I might say that her suit against the street car company is not yet settled; I believe it is to come up in a week or two. That is one factor. Another factor is that ever since the age of seventeen (three years before the street car accident) she had been having these attacks of apprehension. She was a neurotic individual, at least at seventeen, and to say that her trouble at the present time is due exclusively or essentially to the accident is, of course, folly. It is true to the extent that it acted on an unstable individual, and the street car company is liable in that they allowed this unstable individual to ride, but they cannot be ruled off the cars, and if an accident happens the street car company is held up, which does not seem altogether right.

I have said this patient's symptoms are due to traumatic neurosis. A more detailed consideration of the various etiologic and clinical factors will help toward an understanding of the mechanism and development of traumatic neurosis. To an extent this is also true of the mechanism and development of neurastenia.

Etiologic Factors.—First of all the etiologic factors must be considered, and first of these is the material upon which the etiologic factors had to work—namely, the individual.

Up to the age of fifteen or eighteen the average individual has no responsibility. The aspect that he presents to the world at large need not concern him to any great extent. His mistakes

are all condoned with the remark "He's just a child," but after the period of puberty, with its physiologic stress and strain, there comes a dawning consciousness of one's responsibilities, an appreciation of the way in which one must react to one's environment. This environment includes, in my understanding, not only the external world, but also one's thoughts concerning the external world. Up to the time that this appreciation of our duties begins to dawn upon us our desires have full sway. Their only restraint is one of fear of punishment. With this appreciation of the fact that we no longer are irresponsible, and will no longer be excused, there comes a feeling of the necessity of standing well in the eyes of others. This is soon followed by a desire to stand well in our own eyes. The effect of this social desire, if it may be so called, is to make us submit our desires to a scrutiny they have never before undergone. We begin to question our motives and attempt to shape our conduct to fit our gradually developing ideals.

As I have said, this strain comes soon after passing through the strain of the period of puberty. It is easily understood that this checking of our desires, this constant directing of our conduct is bound to be somewhat wearing, is bound to be somewhat of an effort. The desires which we have had and given way to in the entirety of our experience up to a date still, of course, persist. Many of them are recognized as shameful, or as something we do not wish to be suspected of. They are desires possessing their attractive strength in spite of the recognition of their unworthiness. It is this continuance of the desires in spite of the recognition of unworthiness that makes trouble.

Briefly, this is a statement of the action of the various forces that combine to bring about the onset of the neuroses, at least their first appearance, during the years between fifteen and twenty.

You will remember that this girl's first attack occurred at the age of seventeen. It is at present her proud boast that she is a Sunday-school teacher. You will find as a usual thing that the religious impulse becomes strong some time during the second half of the second decade. This patient's turning to religion

occurred at just about the same time that her apprehensive attacks began. As the Bible says, there is just one strength upon which all miserable sinners can depend and that strength is Divine, so in the confusion arising from the first attempts at going out into the world this Divine strength is the one that is turned to, hence the beginning of the religious impulse at this time.

The point I wish to emphasize, the one which bears on this case in particular, is the fact that the religious impulse shows that this individual felt a need for some omnipotent force to come to her aid at that particular time. In other words, her problems were too much for her to handle herself.

This is the ground upon which the subsequent etiologic factors had to work. The apprehensive attacks which began at about seventeen directed her attention from her mind to her body; that is, the physical expressions of fear became the evident and impressive factors rather than the confusion that existed in her mind.

These apprehensive attacks and their physical symptoms emphasized to the poor girl her inability to meet the world on anywhere near an equal basis. Then at the age of twenty, after she had tried her best for three or four years, she suffered this accident. Never since then has she regained her courage sufficiently to take up the fight again.

It is manifestly impossible for anyone to give up and simply say in extenuation of their giving up that the struggle is too much. There must be physical symptoms to furnish an excuse for the capitulation. This girl has never felt right physically since the accident. It has been either a bearing-down, dragging pain in the back, a headache or light-headedness, nausea, or just plain nervousness. There was always some physical complaint.

In consideration of the accident itself we must appreciate the fact that it not only forms an excuse for giving up, but it also acts as an incitor to a feeling of revenge, and there is also the chance to capitalize it—the possible award of damages. Each time I have had any prolonged conversation with the patient the fact that she was expecting the trial to come up at any time has

been mentioned by her. When one sees her efforts to produce vomiting it is impossible to feel that they are entirely involuntary and unconscious. Nausea and vomiting have been complaints ever since the accident. Of course they would mean a great deal in the minds of a jury attempting to decide responsibility.

I have already spoken of the influence of the operation performed for displacement, and the necessity of her taking care of her aunt's children before she was strong enough.

To summarize: We have a rather unstable individual. Circumstances over which she had no control made it necessary for her to work. Twice, at the time of the tornado and of the accident, she had good reason to be terrified; once she was justified in breaking down, at the time of her operation and because of her labors in her condition of weakness. Those are the essential factors to be considered in the mechanism of the development of her present condition.

The **prognosis** in a case of this kind is not good. The individual was too unstable, to begin with. External conditions, family and financial, are not favorable. First of all, she should be treated as a sick person. She should have rest, massage, hydrotherapy, and liquid diet. She should be given a great deal of encouragement, assured that although she is sick there is nothing essentially wrong with the stomach. She should be isolated, away from other patients, and especially away from her family and friends. After her confidence has been gained she could then be put on a full diet and built up. Her strength once re-established, she would have a much better chance to fight her own battles than she has had heretofore.

It is a great mistake ever to tell these people that they have nothing the matter with them. That only makes it all the more necessary for them to prove that there is serious trouble. Inasmuch as their thought has all been of physical disease, a certain amount of physical treatment is very helpful in the beginning. They know they are sick, and any statement to the contrary simply provokes resentment.

It is very necessary to bring about a settlement of any pend-

ing law suits as soon as possible after the accident. The two years that have elapsed in this case have been filled with one physical symptom after another, all tending to establish in the girl's mind the idea that she is sick. If suits could be settled at once there would be no necessity for the sufferers, the injured, to maintain a condition of suffering over a long period. I do not mean that this is an entirely conscious process by any manner of means. It is something almost if not quite beyond the control of the individual. They have been hurt and somebody must pay. That they have to suffer also never enters the equation.

CLINIC OF DR. ROBERT B. PREBLE

St. Luke's Hospital (at Bedside)

AN ACUTE NEPHRITIS DUE TO TONSILLITIS, WITH THE SUBSEQUENT DEVELOPMENT OF PNEUMONIA

February 19th.

Dr. Preble: This boy has some herpes, is quite pale, and there is some edema. The face looks puffy; the eyelids and forehead seem a little bit swollen, and he has a little herpetic eruption on the chin and about the left nostril. The respirations average 20 to 22 per minute. (To Patient): Are your eyes giving you any trouble now?

Patient: They seem a little weak, but I feel pretty good this morning; I didn't get much sleep last night, though, I was coughing so much.

Dr. Preble: But this morning you feel no pain, no shortness of breath?

Patient: There is a little shortness of breath, but I have no pain.

Dr. Preble: There is no cyanosis, but the hands seem to be slightly edematous. They have been obviously swollen. He has had a universal generalized edema, moderate in degree, universal in distribution. What do you suppose that is?

Dr. S.: Kidney disease?

Dr. Preble: It suggests nephritis; could it be anything else?

Dr. S.: It could be due to the heart, but such an edema would not be expected from a cardiac etiology; it might be due to a combination of kidney and heart disturbance.

Dr. Preble: There is a moderate degree of edema, generalized in distribution, unassociated with any particular dysp-

nea. Even if the patient were dyspneic and cyanotic, there would not be a universal edema from the heart alone. The heart compensation is never badly broken when the patient can lie flat in bed as this patient does. Occasionally there is a broken compensation of very high grade, but without generalized edema. Here there is a generalized edema which is due to nephritis. Only once in a long while is there generalized edema in which changes in the urine cannot be demonstrated. These must be called angioneurotic edemas without there being any obvious explanation for them.

This boy has been in the hospital for four days. He entered with a temperature of 102.4°F., pulse 106, respirations 22. Subsequently that evening the temperature went down to 101° F. and a fraction; the next day it ranged from 100° to 102° F., and yesterday from 101° to 102.6° F. This morning it was 98.6° F. at 9 oclock. The pulse is now running 96 to 98, respirations 20 to 22.

Two weeks ago he had acute tonsillitis and generalized pain, which lasted four or five days. The throat remained sore until two days before he entered the hospital. A week ago his face and legs began to swell; he described his condition as "miserable," but he had no pain. He was up and around until about four days before he came into the hospital. There was then acute tonsillitis and subsequently the development of edema, but why the temperature which he still has?

Dr. F.: The herpetic eruption would make one think of a pneumococcic infection.

Dr. Preble (to Patient): How long ago did that eruption come out on your chin?

Patient: About two or three days ago.

Dr. Preble: I do not remember seeing it there yesterday and it looks fresh to me. There is still increased temperature; except for the single practically normal temperature this morning, the patient has had increased temperature constantly, 102° F. and a fraction at one time. Usually tonsillitis lasts only a few days, and, even if there is a nephritis, there is no increase in temperature.

On inspection of the chest, edema appears very definitely over the sternum. There is some enlargement of the cervical glands and of the right sternomastoid just below the jaw; the teeth need some attention, but they are not very bad; the thyroid is not enlarged. The border of the sixth rib marks the lower border of the right lung; the left is at the lower border of the sixth also. The right border of the cardiac dulness is at the right border of the sternum and the upper border is at the lower border of the third rib; the left border is in the fifth interspace. As for the lungs themselves, at least anteriorly, there are no abnormalities on percussion. Dr. F. could not hear any sharp apex-beat of the heart. It is a little diffuse, but I think it is stronger in the fifth interspace. Nothing abnormal can be heard on auscultation. The edema is slightly more marked over the posterior than over the anterior part of the body. It is fairly well marked in the back, but yet not extreme. It is not easy to outline the dulness anteriorly; it is irregular in shape and involves the bulk of the upper half of the right lower lobe. Over this râles may be heard, and right at the end of the scapula there is bronchial breathing—a pneumococcic focus in the upper portion of the right lower lobe. What is the relation of the pneumococcic infection to the nephritis?

Dr. F.: The pneumonia may have begun after the nephritis.

Dr. Preble: During the time he has been observed he has had the temperature, but we don't know how long this focus of infection has been present. What about the relation between pneumonia and nephritis?

Dr. F.: I think it is rather common.

Dr. Preble: Not at all. If the finding of casts in the urine is considered a nephritis, then pneumonia with nephritis is common—but the finding of casts in the urine does not constitute nephritis. It is exceptional to find real nephritis in connection with pneumonia.

Returning to our examination, we note a moderate degree of edema of the feet and legs. The tonsils are greatly enlarged and acute inflammatory changes are still present in both.

The laboratory reports that the urine contains albumin,

frequent small hyaline and granular casts, but no red blood-cells. Its specific gravity is 1020 and it is acid in reaction. Another specimen, a twenty-four-hour one, was 600 c.c. in volume, its specific gravity was 1024; albumin was present; there were frequent granular casts and a few red blood-cells. A functional test with phenolsulphonephthalein gave 30 per cent. returned in two hours, about half what there should be. Examination of the blood showed 4,600,000 red blood-corpuscles, 18,500 white cells, and 72 per cent. hemoglobin. The blood-pressure was 162 systolic, 96 diastolic.

The probabilities are that this disease is primarily an acute nephritis, not an exacerbation of an old one. It was probably due to an infection from the tonsils. Some time between the initial attack of tonsillitis and his entrance here there developed a pneumonia of the right lower lobe. What is the prognosis?

Dr. L.: Pretty good, I think; most of these patients get well, but if the condition becomes chronic the prognosis is bad.

Dr. Preble: Yes, then it becomes the prognosis of an ordinary chronic nephritis. The patient has been in the hospital about four days, and I believe he is getting well so far as the pneumonia is concerned, but the end is coming by lysis instead of crisis. The temperature will probably go up again this afternoon and then tomorrow go down to normal for good. Do you think it is possible that the presence of pus in his pleural sac causes the irregular temperature?

Dr. L.: I would not think so. It could hardly be with these findings; there is bronchial breathing, which speaks against the presence of pus in the pleural sac.

Dr. Preble: Is bronchial breathing heard in patients who have fluid in the pleural sac?

Dr. L.: Yes; sometimes it may be heard above the fluid.

Dr. Preble: We heard it best in the upper portion, did we not?

Dr. S.: The bronchial breathing was above the dulness. Over the dulness I thought I heard some râles.

Dr. Preble: What do you think about that, Dr. R.? Could pus be there?

DR. R.: Pus could be there, but the course of the disease has been so short that pneumonia is the more likely cause of the finding. There should be pain if pus is present.

DR. PREBLE: Pneumonia is more probable than empyema, but in many cases of empyema there is no pain. The greatest reason for thinking that the trouble is in the lung rather than in the pleural sac is that it seems to be too deep.

Treatment.—For acute tonsillitis the patient should have a cleansing gargle. I would not favor having the tonsils removed as long as the pneumonia is still active.

Of course, one must be sure that whatever is done for the pneumonia does not aggravate the nephritis. Phenols are not given to pneumonia patients quite so much now as formerly, perhaps, but I would not give them to this patient, as they would probably irritate his kidneys. So far as the pneumonia is concerned, it may be left alone. One or two points should be kept in mind. It is important that patients with pneumonia have plenty of fluid—plenty of water—and yet in this lad with a nephritis the thing to do would be to limit his fluid.

We have not had to consider the use of morphin with this patient, for he gets rest enough without it, but if he were sleepless I would not hesitate to give him morphin. Nephritics must be watched closely when they are given morphin. It should be used in smaller doses and more frequently than in ordinary cases. There is a tendency for it to accumulate in nephritis, and intoxication occurs from doses which ordinarily should not give any.

In treating the nephritis, hot packs favor elimination. We need not hesitate to put the patient in a hot pack when pneumonia is a complication merely on account of a temperature of 103° or 104° F. Even without a pneumonia, however, we are always a little shy about a hot pack, for every once in a while the heart goes to pieces. Here we have a pneumonia, and one of the great dangers in pneumonia is that the heart will give out. The patient also has nephritis, and we must consider all these points. The heart always must be carefully watched while giving a hot pack. We have been giving this boy hot packs; he has had two and will have another one today. He stands them all right and

gives a good reaction. He had a thirty-minute hot pack yesterday and one the day before. Shall we continue them?

Dr. B.: I do not think there is any contra-indication; by increasing the elimination through the skin the work of the kidneys is relieved.

Dr. Preble: What is eliminated through the skin?

Dr. B.: Principally water and salts.

Dr. Preble: Very little organic material is eliminated through the skin. What is the use of the hot packs if nothing is eliminated but water?

Dr. M.: I thought other things were eliminated as well.

Dr. Preble: A moderate amount of salts and a moderate amount of urea are also eliminated.

Dr. M.: Some fluid is eliminated by the sweats, and there is an indication for increasing his intake of fluids.

Dr. Preble: Why do you wish to increase the intake of fluids?

Dr. M.: On account of the pneumonia?

Dr. Preble: We have decided that we cannot increase the intake of fluids without waterlogging the patient, and the object of giving him the fluid is to wash out material through his kidneys. However, it is a customary and an apparently useful thing to do—get rid of the edema. What else shall we do?

Dr. L.: Give him cathartics.

Dr. Preble: He has been getting some compound jalap and that acts pretty well. Would you do venesection? You know we do take blood from patients with pneumonia frequently. Venesection is very frequently done in pneumonia and much more frequently in nephritis, and much more toxic material is eliminated in that way in a minute than can be secured in any other way in days. This boy has so little evidence of any intoxication that there is really no reason for bleeding him, but it is often a useful thing to do, and if this lad showed any more evidence of uremia we would do it at once. There is nothing that can be done for uremics that compares with venesection. It should not be reserved for a last desperate measure, but should be done as soon as definite evidences of uremia appear. Not so very long ago a boy entered the hospital with acute nephritis and

very soon developed uremic convulsions; we did a venesection, and he regained his consciousness in a very short time.

We will keep this boy in bed, limit his fluid intake somewhat, give him a hot-pack once a day, and a little compound jalap powder once a day. As a rule, we do not pay much attention to the diet in pneumonia; if the patient wishes to eat, food is given, but we do not force feeding, and as a rule the patients do not care to eat much. But with nephritis the matter of diet is important. What would you let him have?

Dr. L.: Some milk and cream; no meat extracts or anything like that.

Dr. Preble: No, the old ladies can't bring him broth. We will let him have milk and cream and cereals and vegetables, fruit juices, and stewed fruit. He is getting all these things, but his appetite is poor and he does not care for anything but the milk and cream and cereals. Within a day or two, when his temperature goes down, he will want more to eat, and it will not be necessary to restrict his diet further. I think he will be very much better within a few days and we will probably see him again.

February 23, 1916.

Dr. Preble: The other morning (four days previously) when we saw this boy his temperature was normal for the first time. That evening it went up to 101° F., but on Sunday the highest temperature was 101.6° F.; yesterday it was 98.6° F. and this morning it is down again. You will remember that we were not able at that time to pick out the course of the tendon sheaths in the hand or the bones in the wrist on account of the edema, but now we can pick them all out and also the costalsternal junctions. There is still some edema about the patient's face, but much less than there was, and he feels much better. The urine, however, still contains albumin and casts.

The patient will probably recover completely. In a few days we will take out his tonsils for him and he will probably be all right. Suppose you see if the lung has cleared up.

Dr. M. (after percussing the chest posteriorly): It still seems dull.

Dr. Preble: You listen to it, Dr. L.

Dr. L.: There are some râles, but I do not hear any bronchial breathing; the voice sounds are transmitted, but are somewhat decreased on the right side. The breath sounds also seem decreased.

Dr. Preble: How should it be?

Dr. L.: If there is consolidation the breath sounds should be decreased.

Dr. Preble: The breath sounds decreased, vocal fremitus decreased; what combination of circumstances would give you findings like those?

Dr. L.: Fluid in the pleural cavity.

Dr. Preble: Do you remember when we saw this patient four days ago the possibility of an empyema was suggested—why did we think there was not one? Do you remember about that, Dr. T.?

Dr. T.: I think you excluded an empyema because increased bronchial breathing and increased tactile fremitus were found.

Dr. Preble: Yes. But today we get dulness where there was clearness four days ago—on Saturday. Could the patient have developed an empyema in the meantime; how are we going to find out?

Dr. T.: If the physical signs point to fluid, a needle could be put in.

Dr. Preble: Would the same physical signs be present from a fibrinous exudate? Is there any difference?

Dr. T.: The physical signs would be just the same. If there is pus in the pleural sac it should be drained.

Dr. Preble: Here we have a boy with an acute nephritis. These findings rather strengthen the possibility of irritation of the kidneys from material which he might absorb from his lungs. It is quite likely—if it were not for the nephritis—that whatever physical evidences of an empyema were present, we would say it is probably just a little one and would wait a few days and see how it would continue. In view of the nephritis we will put in a needle and do an exploratory puncture at once and see what the fluid is—if we obtain any. If it is sterile, we will wait a few

days before doing anything more. (*Instructs intern to prepare needle for puncture.*) The nephritis is still active, but the patient is much better in every way than he was; he is having no headache, and while he looks rather anemic he has a pretty good percentage of hemoglobin. He has been having some increased temperature, but not much—the highest point yesterday was 100° F. The last leukocyte count was 12,000. Can the kidneys ordinarily be felt in a nephritis?

DR. M.: I do not think so.

DR. PREBLE: As a rule, the kidneys cannot be palpated in a male; in a female probably 50 or 75 per cent. can be palpated. Occasionally they may be palpated in a male, but as a rule the amount of swelling is not so great that the kidneys can be palpated. Most likely in this case the nephritis was caused by the tonsillitis and the pneumonia developed after the nephritis. This is not an ordinary combination; it occurs occasionally, but is quite unusual. Of course, frequently albumin and casts are found in the urine in cases of pneumonia. The question sometimes arises, Where shall we stop calling the urine the "cloudy urine" of pneumonia and begin calling the case "nephritis"? Albrecht adopts the arbitrary rule that if the albumin and casts persist for twelve days after the crisis the condition is a nephritis; if albumin and casts disappear before that time it is not a nephritis. Of course such a statement is a mere rule of thumb and means nothing. In other infection, scarlet fever for instance, where nephritis is a common complication, it is much easier to diagnose than in a pneumonia where it is rare. Is there any way to demonstrate whether his pneumonia and nephritis are both due to the tonsillitis? If we take his tonsils out and make cultures from them we are apt to get a pneumococcus, for almost everybody has them. The herpetic eruption on his face points toward the pneumococcus, and it may be that we will get something out of the pleura which may give the organism. No blood-cultures have been made.

A CASE OF TRICHINOSIS: TRICHINÆ DEMONSTRATED IN THE SPINAL FLUID

Dr. Preble: Here is a girl who has been in the hospital not quite two weeks, this being the twelfth day. When she came in she had a rectal temperature of 101° F., which in the evening reached 101.2° F. The next day the temperature was 102.6° F., pulse 90, respirations 22; the third day the temperature was 102.4° F., maximum, 100.8° F. minimum; the pulse was about the same in rate; the temperatures are all rectal temperatures. The temperature recorded was similar for the next thee or four days, so that at the end of the first week the temperature was still 101.8° to 102.4° F. The pulse and respiration were also similar to those mentioned. On the eighth day for the first time the temperature was below 100° F.; on the ninth day it was 99° F., and this morning, the twelfth day, for the first time the patient has a normal temperature. During the entire period that she has been here she has had temperature above the normal, which has gradually fallen from 102.5° F. to a normal temperature this morning. This has all been charted and the chart shows the gradually decreasing curve. What would you think of this case?

Dr. H.: The temperature resembles a septic temperature, but it is not very high. It resembles that of a more or less limited process.

Dr. Preble: All that we know is that this patient has been having a temperature that has been tending downward for at least twelve days. There have been no gross irregularities in it. It has been practically a continuous temperature, but with a downward trend.

Dr. H.: It looks like that of a case of typhoid.

Dr. Preble: The pulse-rate is a little higher than would be expected; it has been 120 on several occasions. When we consider her history we find it exceptionally interesting.

On November 7th she felt very badly, did not eat; was nauseated or "sick at her stomach," and vomited what she had eaten the previous evening. The vomitus was pretty sour and bitter. She did not go to school the next day, but rested on a couch and did not eat until evening. She went to school the remainder of the week, but describes her condition as "miserable." On Friday, November 12th, she became sick, awakened from sleep with pain in her stomach and vomited; this continued all day Saturday with diarrhea. She was sick for about ten days at home and then came to the hospital. On November 12th, the day on which she says she was taken sick, although actually she had been sick all day the previous Sunday and had felt badly all the previous week, her mother noticed puffiness of the eyelids. This grew worse and was present for about a week. On November 14th also she developed a cough which was painful. Her temperature averaged 102° or 102.5° F.

This patient had the ordinary diseases of childhood; has not had typhoid, pneumonia, scarlet fever, or rheumatism. She is fourteen years old, a school girl in the eighth grade, and her habits are just about those of an ordinary girl of that age. To sum up, the history covers a period of about four weeks in which the patient felt badly and had no appetite. After the onset of the disease she got up and went to school for a week. She then had some vomiting and diarrhea, and a few days afterward had fever and general soreness. There developed a cough and puffiness about the eyes. The patient then became more indisposed, went to bed, getting worse for about ten days, and then came to the hospital. As nearly as we know she has had a temperature continuously since about the 14th of November; that is, for about two weeks of November and the four days of this month. What are the possibilities?

Dr. S.: The onset, which was rather slow and insidious, suggests typhoid. She had the gastro-intestinal symptoms, the pain and diarrhea and vomiting.

Dr. Preble: Yes, and she had also a slight cough, muscular soreness, and a continuous temperature. How does it look to you, Dr. M.?

DR. M.: So far as the temperature is concerned it resembles typhoid.

DR. PREBLE: Yes, the temperature has gradually gone down like that of a typhoid and the patient's general condition has improved. Now let us remove her gown and see what we can find. (To Patient): Why don't you put that arm out?

PATIENT: It hurts me so much.

DR. PREBLE: Do any other muscles hurt?

PATIENT: All over my muscles hurt. My back hurts so bad, and my arms.

DR. PREBLE (feeling abdominal muscles and muscles over the thighs): What do you think about that, Dr. M.?

DR. M.: The puffiness of the eyelids and the tenderness of the muscles might make one think of trichinosis.

DR. PREBLE: What have we heard so far that does not fit with a diagnosis of typhoid?

DR. M.: In typhoid, as a rule, there is no puffiness of the eyelids.

DR. PREBLE: Puffiness of the eyelids does not occur in an uncomplicated case of typhoid. As we consider her condition today with the possibility of typhoid in mind, what is inconsistent?

DR. L.: The muscular pain is not the same as in typhoid.

DR. PREBLE: It is out of all proportion. Typhoid patients feel sore and lame, as does anyone who has been through a severe infection, but patients with typhoid do not have the tenderness to pressure that this child has (*patient complains bitterly of pain as Dr. Preble touches different muscles*); she is sore to just the lightest contact over the muscles of her thigh and diaphragm. Whether or not the spleen was palpable at the time when she had her highest temperature I do not know.

PATIENT: The doctor felt for it every day, but he never could find it.

DR. PREBLE: Did he ever make a Widal test? Did he take any of your blood and examine it?

PATIENT: Yes, he took some blood out of my ear and took it to the laboratory, but they said it was not typhoid. As I had been sick only a week the laboratory said that it might not show.

INTERN: The patient did have some rose spots.

PATIENT: One day the doctor found nineteen red spots and the next day he couldn't find any.

DR. PREBLE: When the patient entered the hospital the examination of the urine was negative and the Widal was negative. When the blood-count was made there were 21,000 leukocytes. What does that mean, Dr. L.?

DR. L.: It was very much against typhoid.

DR. PREBLE: Yes, Does not the fact that a man who knows how to feel for a spleen, but fails to find it palpable, speak against a diagnosis of typhoid? The patient entered the hospital with numerous symptoms which speak against a diagnosis of typhoid; what are they?

STUDENT: The pulse was rapid; the spleen could not be felt; there was a leukocytosis and extreme muscle tenderness.

DR. H.: The puffiness of the eyelids would speak against a diagnosis of typhoid.

DR. PREBLE: Yes. The absence of the eruption when the patient entered the hospital and the negative Widal also speak against it, but no one of the symptoms mentioned excludes such a diagnosis. The accumulative evidence against typhoid, however, is very great. If not typhoid, what is it?

DR. H.: I would think of typhoid first and of trichinosis next.

DR. PREBLE: Most suspicious is the circumorbital edema; what does it suggest?

DR. H.: Nephritis.

DR. PREBLE: The urine examination was negative. The patient had a little sore throat before she came in, so she might have had an acute nephritis, but that was excluded readily enough by the fact that the urine was normal. I should think the next thought might be a trichinosis.

DR. M.: The puffiness of the eyelids would come earlier than the tenderness of the muscles.

DR. PREBLE (to Patient): Did your eyes appear blood-shot when you first became sick?

PATIENT: It was red all under here (indicating lower lids).

DR. PREBLE: How are you going to make the diagnosis?

DR. M.: Make a blood-examination, and take a piece of muscle for examination under the microscope.

DR. PREBLE: What do we find on such examination?

DR. M.: There is usually an eosinophilia with a leukocytosis of 15,000 or 20,000.

DR. PREBLE: The blood-count of this patient showed 21,000 leukocytes and she has 21 per cent. of eosinophils. A piece of the muscle was taken out, but the results of this examination are not yet available. Dr. Elliott suggested that we might find something in her spinal fluid. Yesterday a spinal puncture was made. The cerebrospinal fluid was perfectly normal in appearance, colorless, clear and free from cells, but with one trichina embryo to every drop of fluid. I never saw that condition before. Since we found the trichina embryos in the spinal fluid yesterday I learned that Dr. Bloch found them in a similar case at the County Hospital. In the meantime he had looked up the literature and found reference to one other case. This was an entirely new finding to me. It means that we will have to examine the spinal fluid of all of these cases.

The following are the essential points in the diagnosis—the cough, the gastro-intestinal disturbance, the diarrhea, the puffiness about the eyes, the temperature, and the muscular tenderness. The symptoms are not always so well developed as in this case. Cases of trichinosis are far more common than is popularly supposed. Several years ago, at the County Hospital, a record was made of all the trichinosis found at postmortem examinations. About 20 per cent. of all the bodies examined showed that they had had trichinosis. Considering how comparatively few cases are diagnosed, there must be many that are entirely unrecognized. This patient was first told that she had grip. For example, a patient may give the following history: He has been feeling sick, stiff and sore for a few days, and then all his symptoms disappeared. Such an experience is common. There is nothing about it that particularly suggests a diagnosis of trichinosis. Two or three members of the same family have similar symptoms; then one of them develops a swelling about the eyelids and is taken to a hospital. With the other members

of the same family in mind, a blood-count is made and with it a diagnosis of trichinosis.

A few months ago I saw the following case: A girl entered the hospital with a little swelling under her eyes and no other symptoms. Her attention had been called to a little puffiness under the eyes. We made a blood-count and found-about 7 per cent. of eosinophils, and this went up until she had 20 per cent. Still she has no other symptoms—no gastro-intestinal disturbance, no cough, no nausea. It is therefore easy to see how trichinosis can be overlooked. When circumorbital edema appears, the thought should always be, "That may be trichinosis." Whenever you see a case that resembles typhoid, make a blood-count. Then your diagnosis is relatively simple.

The circumorbital edema usually lasts not more than three to five days. With it there may be a good deal of conjunctivitis and subconjunctival hemorrhage, and at that period there may or may not be an increased temperature.

As to where this girl got her trichinosis: On the 31st of October she and her family ate some pork chops and the next day she had some roast pork. None of the other members of the family were taken ill. This girl also had some pork chops a few days before with a little girl friend, and that girl is ill, but we have not been able to find out whether she has trichinosis or not.

Not much can be done in the way of therapy. As the soreness of the muscles is a myositis, it might be relieved with salicylates. The salicylates should be used in small doses over a long period of time.

It is impossible to prevent the invasion of the trichina. One does not know the condition is there until it develops. Clinically we do not know anything about it until after the invasion. If the patient has symptoms after eating pork, they are, as a rule, just the usual gastro-intestinal symptoms that come from a number of different disturbances. If a whole family gets sick after eating pork an examination of the pork might reveal the organisms, but usually the physician does not learn of the condition early enough and the cases are not seen until the embryo has developed and migrated. The development and migration

are accompanied with so little irritation that the physician hears nothing about the symptoms. This girl had her pork some time before the 1st of November, and on the 7th she felt sick, but she dates her illness from the 14th. There was, therefore, a period of two or three weeks before she got so sick that she had to go to bed; then came a period of observation during which it was uncertain what the trouble was. Then suddenly appeared the phenomena that are associated with the wandering of the embryos, the irritating phenomena as they pass through the muscles.

The intensity of the myositis varies greatly. In one case which I have mentioned there were no symptoms except that the patient felt a little stiff, lame, and sore when she got up in the morning. The pain ranges all the way from that described to the most intense pain—sometimes $\frac{1}{2}$ grain of morphin has no effect on the pain. The muscles around the attachment of the diaphragm are particularly liable to be affected. The parasites tend to settle also along the tendinous ends of the muscles and the soreness is intense. Along with these phenomena are those of infection—a temperature which usually does not last more than ten days, sometimes less than that, sometimes lasting for two or three weeks, and occasionally running as high as 104° or 105° F. Increased temperature usually does not continue very long before it becomes grossly irregular. The physician thinks "that's a funny typhoid," but the simulation may be very good. The pulse is relatively slow, there may be an eruption which resembles the rose spots of typhoid, and the case may actually appear to be typhoid until the blood-count is made. An eosinophilia may be found in the counting chambers—you don't have to make a stained specimen to find them. A leukopenia is found, and occasionally embryos are found in the blood, although they are very hard to find. Another early symptom, and one that is very peculiar, is the circumorbital edema and the subconjunctival hemorrhages. When such an edema as that is present in the course of the temperature the possibility of a trichinosis is at once suggested, particularly as the patients usually have a normal urine. For a varying period, usually a few weeks, the disease continues and then becomes quiescent.

These patients sometimes die of the disease. There have been outbreaks, particularly in Europe, where the mortality has been very high. There the cases are more likely to occur in groups. Because of the manner in which our meat supply is handled we do not often see epidemics of it in this country. Here a piece of the same hog may go to one customer in Chicago, another piece to Ohio, and another to South Dakota. We do not have the epidemics that appear in those countries in which the hog is slaughtered and distributed to the people in the same neighborhoods. So far as my personal observation is concerned, I have never seen a fatal case and have seen none who were ever ill for more than a few weeks. This may be due to the fact that in this country people rarely eat their pork uncooked, while in many places in Europe many of the people eat it raw.

A few months ago I saw the family of a German physician. All the members were very fond of raw pork; a few days after eating some of it the wife developed a beautiful "muscular rheumatism"; the husband had some stomach trouble, but was not very sick, and the cook who did not cook the pork was somewhat uncomfortable. The wife was very sick; she had a high eosinophil count, and the husband, who was not very sick, had 56 per cent. of eosinophils; the cook had 57 per cent. The eosinophilia is sometimes as high as 80 per cent., but I have never seen one that high.

DR. L.: Would a catharsis do any good?

DR. PREBLE: If the patient is seen early enough catharsis may do good; later it is of no service. The trichinæ multiply in the intestinal tract, then the embryos begin to wander, and the trouble begins. When we make a diagnosis of trichinosis in a patient we always clear out the gastro-intestinal tract, but the trouble has already started.

DR. L.: I meant the use of the anthelmintics.

DR. PREBLE: Such treatment will not shorten the course of the disease, but it may keep other embryos from developing. It is best to cleanse the gastro-intestinal tract thoroughly with calomel and salts.

INTERN: How about neosalvarsan?

DR. PREBLE: I do not know about its use in this disease.

INTERN: Dr. F. G. Harris has published an article to the effect that neosalvarsan is just as effective in these cases as in lues.

DR. PREBLE: I have not seen it—it is something new. It is interesting to see these cases, and having seen them you are not likely to overlook them. The great majority of cases of trichinosis are overlooked because physicians have not seen cases of the disease.

CLINIC OF DR. FREDERICK TICE

COOK COUNTY HOSPITAL

PLEURISY WITH EFFUSION. THE INDICATIONS FOR THORACENTESIS AND ITS TECHNIC IN DETAIL

THIS man, who entered the hospital on the 14th of December, is thirty-nine years of age, a barber by occupation, and gave this history: The onset of his illness occurred three weeks ago, consisting of pain in the region of his heart, which was aggravated by deep inspiration and which radiated into the back. It persisted for one week. At times the patient felt definitely chilly and at other times hot and feverish. Two weeks ago he visited a dispensary, where he was told that his temperature was 102° F. and was given some medicine. One week ago, at a subsequent visit, his side was strapped, and at a still later visit an aspiration of his chest was done and half a pint of fluid removed. He felt better after the aspiration. He has had very little cough, no expectoration, and no sweats. He had pleurisy one year ago. He has a skin disease of fourteen years' duration which the dermatologists say is psoriasis. For the last two months this cutaneous involvement has been much worse.

Family History.—The father and mother died of old age.

Personal History.—He had a specific infection seventeen years ago; has used alcohol rather to excess, particularly during the past three months.

When this man entered the hospital his temperature was 99.4° F.; four hours later, 99.8° F.; at midnight, 99° F.; the following morning, 99.4° F. It has remained between 99° and 100° F. since his entrance. The pulse has ranged between 90 and 110, respirations between 20 and 24. The patient is able to assume the recumbent position without any discomfort. Upon exam-

ining the chest we find that there is a difference between the two sides. The left side is larger than the right; there is a fulness particularly in the precordial region; the left clavicle seems higher and the infraclavicular space is more filled out than the corresponding side. There is no discomfort on forced breathing and the amount of expansion is apparently about equal. There is a good diaphragm phenomenon on the right side, but no shadow on the left. The apex-beat is not visible; there is an indefinite pulsation, but no localized, circumscribed apex-beat. On percussion, with the patient in the dorsal position, there is good resonance over the entire left chest anteriorly. The resonance on the left, as compared with the right, is relatively dull, but it is not flat; Traube's space is present. The right border of the heart is at the right border of the sternum, the base at the lower border of the third rib; the left border just inside the nipple line. With the patient in the upright position, on percussion anteriorly over the left side we find the note in the infraclavicular area materially changed. In the recumbent position there was slight dulling of the percussion note, but in this position there is hyperresonance, and as we percuss downward we encounter dulness which becomes practically flatness, and Traube's space is obliterated. Posteriorly, first on the right side, the percussion resonance is normal, extending for a hand's breadth below the inferior angle of the scapula; on the left side there is relatively a hyperresonant note above, and as we percuss downward the note becomes gradually more and more impaired, so that on a level with the inferior angle of the scapula it is flat and there is a marked sensation of resistance.

On direct percussion there is not only a dull note but also a sense of resistance. A paravertebral dulness or Grocco's triangle is present on the right side. On palpation, tactile fremitus is absent over the area of dulness, and on auscultation the breath and voice sounds are both absent.

The diagnosis can be made at this time of fluid in the chest. Recalling the physical signs found in such a condition, we have, first, flatness on percussion, with a marked sense of resistance; second, the absence of tactile fremitus; third, the absence of

breath and voice sounds; fourth, movable dulness on change of posture; fifth, displacement of neighboring organs; and, sixth, one of the cardinal points, the aspiration of fluid by puncture. This man has been aspirated twice, the first time in the dispensary, when half a pint of fluid was removed. He has been tapped once since he entered the hospital, but the result of that thoracentesis is not recorded on his record.

THORACENTESIS

Earlier in the year we showed you a patient with a pleural effusion on whom a thoracentesis was done here in the clinic. We did not at that time go into the details of thoracentesis, and we have not yet given the subject sufficient attention. When is a thoracentesis indicated? When are we to know that a puncture is required and an aspiration indicated, and when is it best to leave the fluid alone? There are three very definite indications. The first is the so-called vital indication; that is, when there is danger to the life of the patient. This may exist when there is relatively little fluid in the chest, when there is shortness of breath, dyspnea, and cyanosis, when the pulse is rapid and irregular, and when the patient is in a cold, clammy sweat; that is known as the vital indication.

Second, there is the so-called *Trousseau's indication*, when there is an enormous amount of fluid in the chest, not necessarily accompanied by symptoms. This was the condition of the patient referred to, the patient who died suddenly with an enormous pleural effusion on the left side without having had any discomfort whatever. The fluid extended up to the clavicle, and on autopsy an embolism of the pulmonary artery was found. In the experience of a number of clinicians sudden and unexpected death may occur where one pleural cavity is nearly or completely filled with fluid.

Fräntzel relates that on making his rounds at midnight, he found a patient in the ward with an effusion completely filling one side, and he made a note that aspiration was to be done in the morning. But before morning the patient was seized with a sudden attack and was dead before assistance could be rendered.

The third indication is delayed absorption; that is, if after waiting a certain period of time the fluid does not disappear. Traube and Bowditch said that paracentesis should not be done before the third week; Fürbringer stated that three or four weeks should elapse, and Weber favors waiting fourteen days, but believes that some cases should be aspirated on the sixth or eighth day after the effusion has accumulated. On the whole, the latter part of the third week is generally accepted as the proper time for doing a paracentesis.

One-third of the cases of effusion recover without operative treatment; the remaining two-thirds require aspiration or rib resection. The reported cases are not very abundant, but they show that in about 75 per cent. of the cases aspiration is done for delayed absorption. The present tendency is to avoid long delay; aspirations are being done earlier than formerly. The vital indication exists in about 20 per cent. of the cases and Trousseau's indication constitutes the remaining 5 per cent.

Method of Procedure.—The position depends upon the condition of the patient and the wish of the operator, but, generally speaking, the patient should be placed in an upright position and, if possible, on a chair or stool, with the head and shoulders resting on a pillow on the back of another chair, with the arms elevated so as to increase the intercostal spaces. If this is not feasible, the patient may lie on the edge of the bed with the arm on the involved side placed upon the head.

The Preparation of the Patient.—The usual tincture of iodin preparation should be employed or any other desirable method of producing a surgically clean site for the puncture.

The Site of the Puncture.—This is not always a matter of choice; if the fluid is encysted, the puncture must be made over the site of the findings. Otherwise the puncture may be made in one of a number of locations. The site recommended varies according to the opinion of the operator. Bowditch and Immerman prefer the ninth intercostal space in the scapular line; Dieulafoy, the eighth intercostal space in the scapular line; Naunyn, the eighth intercostal space posterior to the mid-axillary line; Trousseau, the seventh intercostal space in the axil-

lary line; Gerhardt, the sixth intercostal space between nipple and axillary lines. Probably the site of puncture which is the most popular is that of Dieulafoy, the eighth intercostal space in the scapular line.

There are a few precautions to be observed in making the puncture: First, the puncture must be made in the region where the fluid is located by the physical signs; second, the abnormal or normal position of the neighboring organs must be considered—that is, displacement of the heart, the liver, or the spleen; third, the anesthetic. Ordinarily no anesthetic is necessary, or, at best, a local anesthetic—sometimes ether sprayed at the point of puncture, or ethyl chlorid, or the application of an ordinary piece of ice is all that is necessary.

In choosing the instrument use a small trocar to prevent laceration of the lung. In introducing the trocar into the space selected it is pointed upward and inward, hugging close to the upper border of the lower rib of that intercostal space so as to avoid injury to the artery and nerve.

The question of suction must also be discussed. Fluid in the pleural cavity is usually under a positive pressure and no suction is necessary for its removal. Ordinarily the introduction of the trocar, with a rubber tube attached for removing the fluid by siphonage, is all that is necessary.

The next consideration is the rapidity of the flow. It is essential not to allow the fluid to escape too rapidly, otherwise some of the untoward effects of aspiration may develop. The rule is not to remove more than one liter every twenty minutes.

Next, the amount of fluid to be removed must be considered. Some advocate removal of all the fluid or as much as possible, but a safe rule, and the one generally adopted, is the one made by Fräntzel, not to exceed 1500 c.c. After this amount is removed, if fluid still remains it is better to discontinue the aspiration and repeat it at a subsequent time—if necessary, the same day.

The wound is closed either by adhesive plaster or a cotton and collodion dressing. Repetition of the puncture depends upon the presence of one of the three indications for aspiration.

AN ACUTE PNEUMONIA ADDED TO PULMONARY TUBERCULOSIS

Diagnosis Made by Deduction from the History, Clinical Course, Physical Findings, and Laboratory Analyses. Consideration of Other Possibilities.

January 17, 1916.

THIS patient entered the hospital on December 10, 1915. She is twenty-six years old and gave this history: The condition started five days before entrance, with pain in the lumbar region and the right side of the chest. The pain in the back was of a dull, aching character, and immediately following this the back became so stiff she could not bend it; the pain in the chest was sharp and stabbing, which was aggravated by respiration. This condition continued for two days, when the patient noticed a feeling of chilliness followed by fever. She next began to notice marked shortness of breath and palpitation of the heart. Just preceding her entrance to the hospital these symptoms increased in severity and her hands and feet became swollen. She had no cough.

Past History.—She had the usual diseases of childhood. Six years ago she had some heart trouble, with swelling of the feet and pains around her heart, accompanied by shortness of breath. This has recurred at times in short paroxysms. She had pneumonia followed by empyema three years ago. Since then the patient has never felt well; she has been constipated, her appetite has been poor, and there has been a great deal of weakness and inability to work.

Marital History.—The patient was married three years ago; her husband is living. There was one miscarriage at five months. Her last menstrual period occurred a year and a half ago.

Habits.—She uses wine occasionally. Venereal infection of any kind is denied.

When the patient entered the hospital the temperature was 98.6° F., pulse 84, respirations 24; that was the midday record.

No afternoon record was made, but at midnight the temperature was 101° F., pulse 88, respirations 24. The following morning at 4 o'clock the temperature was 101.8° F.; no noon record was made, and the afternoon record showed a temperature of 104.4° F. The temperature curve on the afternoon of the second day between four and eight o'clock reached 105° F., but the following morning it had fallen to 100.3° F., and that afternoon reached 101.4° F. It fell the next morning, and in the afternoon was again 101.4° F., dropping to subnormal the following morning. Since that time the temperature each day has been approximately normal in the morning, going up in the afternoon usually 1 or 1½ degrees, so that there is and has been continuously a slight pyrexia. On the 29th of December, at eight o'clock in the morning, it was 99.6° F.; in the afternoon of that day it only reached 99° F., but, as a rule, the temperature is higher in the afternoon than in the morning. On the 14th of this month (January) the morning temperature was 100.2° F.; no afternoon temperature is recorded.

On examination we note that the patient is able to assume the dorsal position without any discomfort; there is no dyspnea. The first glance reveals a definitely anemic condition, apparent in the external surface and the mucous membranes. On examination of the chest we observe at once a disproportion between the two sides, the left being much smaller than the right; there is a retraction of the supra- and infraclavicular regions. The right side is full, relatively distended, and on ordinary respiratory efforts there is better expansion on the right than on the left; on forced respiration the condition is much exaggerated; there is a definite lagging on the left side.

On inspection we further note the location of the apex-beat in the fifth intercostal space in the midclavicular line. In a favorable light the diaphragm phenomenon can be seen on the right side, but is absent on the left.

On percussion over the right side the hepatic dulness is found at the fifth intercostal space, the right border of the heart at the right edge of the sternum. There is good resonance over the entire right side; on the left there is dulness and, as you can observe, discomfort—even light percussion produces a wincing

and manifest discomfort. In the axillary region there is dulness closely approaching flatness; it is rather difficult to percuss the left side because of the tenderness, but you can determine the presence of dulness, particularly in the axillary region, where it is almost flat as compared with the right side. It is impossible to determine the exact outline of the heart for the reason that the cardiac dulness merges into the pulmonary dulness.

Posteriorly, on the left side, we at once observe a scar, the site of the operation for empyema three years ago following pneumonia. We also observe that the left shoulder is a little lower than the right, and on forced respiratory efforts there is more expansion on the right than on the left. On percussion there is impaired resonance over the entire left side. On the right side the note is slightly hyperresonant, and extends down a full hand's breadth below the scapula. On the left there is not only the impaired resonance above and below the scapula, but the note becomes relatively flat below; tactile fremitus is present and increased over the right, but relatively decreased over the left, and as we approach the lower portion tactile fremitus is almost gone.

On auscultation both anteriorly and posteriorly the breath sounds are present; over the right side they are slightly roughened, while over the left side they are definitely bronchovesicular, accompanied by numerous râles, most of them subcrepitant in character. This is particularly true of the left upper lobe above and below the clavicle. There is also bronchophony. Over the heart there is a systolic murmur at the base.

The abdominal examination reveals nothing abnormal.

When this patient entered the hospital you will recall that there was a complaint of extreme soreness, and upon the least movement severe pain in the lumbar region, in fact up and down the entire spine. At this time there is no discomfort. The anteroposterior and lateral movements show no limitations of motion and no tenderness or soreness. On the day the patient entered the hospital these findings were recorded: "The right chest expands well, but the left side is greatly reduced and the expansion limited. There is a marked Litten's sign on the right, which is absent on the left. The entire right chest is slightly

hyperresonant, the borders are free, and there is good respiratory excursion. The left chest anteriorly and posteriorly is dull. On auscultation exaggerated vesicular or bronchovesicular breathing is present over the entire left chest. There are numerous fine, crepitant râles on inspiration over the entire left chest. Examination over the heart shows the apex-beat in the fifth intercostal space, rather diffuse, a systolic murmur at the apex and at the base."

With these findings at that time several conditions were considered in the differential diagnosis. There was the history of pneumonia three years ago followed by empyema and drainage for several weeks. The patient was very definite in stating that at the time of the removal of the tube she was not well and that she has continued to be ill off and on ever since. One of the possibilities which was entertained was the existence of an empyema. Was there an old pleural infection dating from the time of the pneumonia and empyema three years ago and continuing up to the present time, with a reaccumulation of fluid, as a cause of the present illness? The physical signs were not suggestive of an empyema. The presence of Traube's space as it exists at the present time, the position of the heart, the presence of bronchial breathing, associated with a decrease in the size of the chest, rather argued against an empyema. But in these old empyemas the signs are often not recognizable, and with diminished or almost absent tactile fremitus and absence of breath sounds in the lower portion of the chest, and with both these signs increased above this area, there was a question of an empyema with compression.

Another possibility suggested itself to us very strongly: We do not know definitely as to the character of the pneumonia from which this patient suffered three years ago. Was it a croupous pneumonia, or was there a pulmonary tuberculosis and empyema? If this condition was primarily a pleurisy, perhaps first serofibrinous and later purulent, with exploratory puncture and drainage, it would be very reasonable to suppose that such a condition continuing over three years might go on and produce not only an adhesive pleurisy, but a pulmonary tuberculosis.

Another possibility was the existence of a pneumonia; that is, three years ago a pneumonia with empyema and drainage, followed by relative recovery, and then a week or ten days before she entered the hospital a reinfection—either the so-called grip infection involving the left side or a pneumococcus infection followed by pneumonia.

There was a still further possibility—a pulmonary tuberculosis —and on top of that an infection, either by the grip bacillus or a pneumococcus or mixed infection.

So the possibilities here were numerous and rather difficult to determine. The white count on the day of admission was 30,500. The urinalysis on the same day gave a specific gravity of 1012, acid reaction, a two plus albumin reaction, with hyalin and granular casts. Subsequent urinalyses on the 13th and 15th of December, and again on the 15th of January, revealed practically the same findings—a moderate amount of albumin with a relatively low specific gravity and hyalin casts in fair numbers.

In order to determine so far as it was possible the presence or absence of fluid in the chest, a paracentesis was made on the 13th of December, and at that time a puncture was made both anteriorly and posteriorly—first behind, just above the location of the scar, and the needle inserted in various directions. The point of the needle was apparently engaged and continued suction revealed absolutely nothing. On the same day a puncture was made anteriorly with the same result—a dry tap. On the 14th another puncture was made posteriorly, again with the same result, so it seemed reasonable to conclude that fluid was not present, either serofibrinous or purulent, and if the temperature, the leukocytosis, and the physical signs were due to fluid then it must be encapsulated. The possibility of a pulmonary abscess or an interlobar empyema which was not detected by the exploratory puncture had also to be considered.

To assist still further in the diagnosis, a blood-culture was made on the 12th of December, but revealed nothing. On the 16th of December another blood-culture was made and revealed the presence of a diplococcus, which was Gram-positive and apparently a pneumococcus. This, then, at least in part, elucidated the diagnosis, and we were compelled to assume the

presence of a pneumococcal infection with a consolidation of the lung probably due to the pneumococcus. But this did not entirely account for the physical signs, nor has it entirely accounted for the subsequent clinical course.

As you have observed from her chart, six days following her entrance to the hospital, the temperature became practically normal, not by crisis but by lysis, and since that time has been practically above normal, a slight morning elevation and a little higher elevation in the afternoon. This patient was sent up to the *x*-ray department for fluoroscopic and *x*-ray examinations, partly for the chest, but more particularly for the vertebral condition. During the first week of her stay in the hospital she could hardly move in bed. By preference she would rest upon one side or the other, rarely lying upon her back, and the physical examination was very difficult because of the severe pain which was produced upon movement. The *x*-ray examination on the 14th of December revealed little or nothing. The report was to the effect that "there is a rather indefinite outline of the eighth, ninth, and tenth dorsal vertebræ, but there is nothing absolutely definite in the fluoroscopic or *x*-ray examinations." The subsequent course would justify us in believing that this vertebral trouble was probably an acute condition associated with her other infection, the mixed type or pneumococcic infection. With the fall in the temperature the vertebral symptoms improved and at the present time have practically disappeared.

What is the condition at present? Is this a delayed resolution following a croupous pneumonia or a mixed infection, or is there a pulmonary tuberculosis? Or is there still some other condition? The examination of the sputum is of the greatest importance. On the 15th of December the sputum was mucopurulent, with many leukocytes present, a fair number of epithelial cells, no elastic fibers and no tubercle bacilli, but there were a large number of pneumococci. A daily order for sputum has existed since that time, but the patient constantly informs us that she does not cough, and it has been impossible to obtain a suitable amount of sputum during all the time she has been here. Yesterday a small amount was obtained, and examination revealed a few red cells, some epithelial cells, and some pneumococci,

but no tubercle bacilli could be determined by the antiformin method. So it has been impossible to determine by the sputum the presence of a tuberculosis, or at least an open tuberculosis.

As far as can be determined from the physical signs when she entered, the subsequent clinical course, the present physical signs, and the laboratory findings, this patient evidently suffered from an acute infection producing the signs of a pneumonia on the left side, which continued for practically a week, and since that time the findings of a pulmonary consolidation involving practically the entire left lung.

At present we can make this clinical diagnosis: A pneumonia probably due to the pneumococcus (judging from the number in the sputum and the positive blood-culture), and at this time a delayed resolution involving practically the entire left lung, with the existence in the lower part of the lung of an old adhesive pleurisy, the result of the empyema.

Etiologically, it is wise to determine the cause of this delayed resolution and whether a tuberculosis is present or not. We believe from the history of an empyema which was opened and drained that a tubercular infection is very likely. The patient says she has never felt well since the time of that infection. It is almost certain that in the course of a few days or a few weeks tubercle bacilli will appear in the sputum.

It is also interesting to note that this patient, a year ago last Christmas, was sent out to the tubercular department at Oak Forrest and was there until the following May. During that time the sputum was examined repeatedly, but no bacilli were ever found. She also tells us that she received repeated injections which she understood were tuberculin, and it is quite safe to assume that clinically, even a year ago, this patient was looked upon as tuberculous. If that is true then we are still more correct in assuming that an old pulmonary tubercular infection existed, to which was added an acute pneumococcic condition. Perhaps the majority of the findings as they exist today are due to the old pulmonary infection which undoubtedly will become worse, as the pneumonia tends to cause an exacerbation. We believe that tubercle bacilli will eventually be found in the sputum.

ACUTE PNEUMONIC PHTHISIS

Frequently Only Suspected When a Supposedly Croupous Pneumonia Fails to Resolve. Tubercle Bacilli may be Found Early in the Circulating Blood. Negative Tuberculin Reaction the Rule in Acute Forms of Tuberculosis, Owing to Absence of Antibodies.

January 24, 1916.

DR. TICE: What is your diagnosis in this case?

VISITING PHYSICIAN: On the left side there is consolidation, with pleurisy; he has tuberculosis and is markedly anemic.

DR. TICE: Did you see the history?

VISITING PHYSICIAN: Yes, sir.

DR. TICE: The diagnosis in this case was by no means easy without a knowledge of the sputum findings. The patient is a Bulgarian, thirty-four years of age, a baker by occupation, who was admitted to the hospital on the 20th of the month. The history, so far as could be obtained, is as follows:

This patient was well until forty days before he entered the hospital, when he was taken with quite a severe chill, followed by fever with marked thirst. Up to that time he had been in good health, but since then he has been constantly ill and has lost 20 pounds in weight. During those forty days he was not continuously confined to bed; he consulted a doctor at his office during the greater part of the first week. Since then he has been in bed from time to time, but he walked into the clinic.

Past History.—About seven years ago he had an abscess in the right side of his chest, which was opened and drained. Whether it was a subcutaneous, pyogenic infection, or a tubercular rib, or an empyema, it is impossible to determine from the information he can give us. Aside from that the patient has had little or no previous illness.

Personal History.—His parents are living and well; one brother and three sisters are living and well. There is no history of tuberculosis or carcinoma on either side of the family.

The day he entered the hospital his temperature in the evening was 100° F., pulse 118, respirations 26. The following morning at eight o'clock the temperature was 99° F., in the evening 101° F.; on the 22d the morning temperature was 98.3° F.; in the afternoon 101.4° F. On the 23d, yesterday, in the morning the temperature was 99° F., in the afternoon 100° F. This morning the temperature is 98.4° F. So during his stay here in the hospital there has been only a slight elevation of temperature, chiefly in the afternoon.

On inspection we find not only evidences of an anemia in the cutaneous surfaces and the mucous membranes, but of a loss in weight, which the patient states is 20 pounds. On inspection of the chest we find a marked difference in the two sides—the left is much smaller than the right; there is a retraction of the supra- and infraclavicular areas, with retraction of the lower portion of the thorax, so that the entire left side is considerably smaller. The right side is full and bulging. There is a difference on expansion even with quiet breathing; with forced breathing it becomes more pronounced—very limited on the left, much better on the right. There is a very pronounced Litten's sign on the right side. On the left side there is a complete absence of this phenomenon. The cardiac impulse, particularly the apex-beat, is not easily located on inspection; a more definite impulse can be palpated in the fifth intercostal space just inside the nipple line.

On percussion there is tympanitic dulness above the left clavicle; when we reach the upper border of the third rib the note becomes flat. Traube's space is present. Comparing the percussion note on the left side with that on the right we find the latter full and resonant.

The right border of the heart is at about the midsternal line. The left border cannot be determined because it is continuous with the pulmonary dulness. Percussion on the right side above the clavicle shows impaired resonance; below the clavicle and over the remaining portion of the lung anteriorly the note is hyperresonant. Percussion posteriorly over the left side of the chest gives a flat note from apex to base; on the right side there is impaired resonance above, but as we proceed downward there

is a hyperresonant note a full hand's-breadth below the angle of the scapula. Tactile fremitus anteriorly and posteriorly over the left chest is increased; there is a relative increase over the right side both anteriorly and posteriorly.

On auscultation, over the left upper lobe anteriorly and posteriorly, the breath sounds are bronchovesicular, accompanied by numerous râles, many of them rather loud and moist in character. No definite click is present. Over the lower lobe the breath sounds are tubular, with only a few râles. Bronchophony is present. Over the right side anteriorly, just above and below the clavicle, slight bronchovesicular breathing and a few râles are found. Over the remainder of the lung auscultation is practically negative. Auscultation over the heart reveals practically normal tones.

Examination of the abdomen is negative; the liver and spleen are not palpable.

So we have as the chief findings the pulmonary signs, which consist of the signs of infiltration or consolidation of the upper and lower lobes of the left lung, with indications of some infiltration of the right apex.

This man was taken sick, as you will recall from the history, forty days before his entrance here. He is very positive about the date of the onset; but in spite of that the physical examination revealed then, as now, a consolidation or infiltration of the entire left lung. Naturally, the question which occurred at that time, and also the question which concerned the attending physician who took care of him before he entered the hospital, was whether this man suffered from an acute pulmonary infection of pneumococcal origin, or a grip infection, with delayed or unresolved pneumonia, or a tuberculosis.

Repeated sputum examinations were made before he came to the hospital, the last one a day or two before he entered, which revealed the presence of tubercle bacilli. That was some days after the onset. He had been sick for three, or perhaps four, weeks before the sputum showed the bacilli. Examinations made since his admission here reveal an excessive number of bacilli; as indicated on the history, he has five plus.

An attempt was made to determine the probable nature of this infection by the use of tuberculin. A von Pirquet was done a few days ago (you can observe the site of this Pirquet on the arm), but there was no reaction whatever. This is of some interest, particularly when we note that this man's sputum is simply loaded with tubercle bacilli. If you do a tuberculin test on a patient in a similar condition and depend upon the result to make or disprove the diagnosis you will be misled. In this case the von Pirquet was absolutely negative, but in spite of that the man has the physical signs of an extensive involvement and the sputum is loaded with tubercle bacilli.

It must be remembered that in acute pulmonary tuberculosis, miliary tuberculosis, and acute tubercular infections of different parts of the body a tubercular reaction is quite frequently negative. In miliary tuberculosis it is the rule to find it negative, due to the absence of the antibodies at the time the test is made.

As to the *clinical course*, we regard this as an acute case. He was perfectly well up to forty days before entering the hospital, when he was seized with a chill followed by a fever, and a cough, with the physical signs of pulmonary consolidation. The condition was looked upon as an ordinary pneumonia. It is true that the patient was up and about, going to the doctor's office to consult him; he subsequently was partially confined to his bed. The physical signs were those of an acute pulmonary consolidation. After a lapse of ten or twelve days, when resolution did not occur and the physical findings did not indicate any tendency to resolve, suspicion was raised as to the probable nature of the infection, and examination of the sputum eventually revealed the tubercle bacilli.

A differential diagnosis between a pneumonia and an acute pneumonic phthisis is often exceedingly difficult. There are some points that may be of assistance. First, if there is a history of pulmonary tuberculosis, particularly in immediate members of the family, it might arouse some suspicion, but in this case there is no such history.

Second. The history of previous disease, possibly tubercular in origin. We have in this case the history of some local infection

about seven years ago; whether that was a tubercular rib or whether it was an empyema we cannot definitely determine. At any rate it would seem to be rather doubtful.

Third. There is really nothing in the onset of the two conditions of any significance. Acute pneumonic phthisis usually comes on with a chill, pain in the side, a sharp rise in temperature, accompanied by a cough, and on the second or third day the sputum becomes rusty, definitely of a pneumococcic type, and the entire clinical course for the first week or ten days is identical with the course of a typical pneumococcus infection. Occasionally the sputum may be hemorrhagic rather than rusty, and if it is examined during the first two or three days, particularly after the fourth or fifth day, the bacilli may be found.

Fourth. The blood examination. The white count usually shows an increase in either disease. A blood-culture is of more assistance, for in a croupous pneumonia, particularly during the first few days, it may be possible to isolate the specific organism. On the other hand, in an acute pneumonic phthisis, if the blood-culture is negative, search for the tubercle bacilli in the circulating blood may be successful.

Fifth. Probably the first indication to cause worry or suspicion that the condition is not the usual pneumococcic pneumonia is the course of the disease—it continues after the day of the expected crisis. Instead of a crisis the temperature may fall by lysis, becoming intermittent in character. This may be associated with pronounced chills or sweating and the physical signs will persist. If the sputum is examined at this time it is usually possible to find, not a few, but many tubercle bacilli.

Sixth. The physical signs during the acute stage of the disease are absolutely identical. All the local physical signs are the same. There may be some difference in the matter of localization. In an acute pneumonic phthisis the upper lobe is more frequently involved. In a pneumococcic infection the lower lobe is more frequently attacked. Of more significance, perhaps, is the occurrence of a consolidation on one side with a slight involvement of the opposite side. In this patient we have one entire lung completely consolidated and with definite physical

findings of involvement of the right apex as well. Thus the physical examination alone would make us suspicious of a tubercular infection.

Prognosis.—Concerning the prognosis, most of these cases run an unfavorable course. The fact that the von Pirquet is negative, showing a lack of antibodies, would at once suggest little or no resistance. After the lapse of four or five weeks, often during the first three or four weeks, these cases go on to a fatal termination. On the other hand, the acute manifestations resembling the acute pneumococcic form may gradually subside and assume the features of the chronic ulcerative type. That would seem to be the tendency in this case. This man has now been ill for forty-three days, with the temperature going down, the pulse considerably less than it was, and the respirations running 24 to 26. So it would seem reasonable to assume, with the indications of a definite softening in the left apex, that perhaps within the next week or ten days physical signs of a chronic, ulcerative type of the disease will appear.

When this occurs the prognosis is that of the usual ulcerative form, with the exception that, as a rule, it is not so favorable because of the lack of resistance. The patient is now being kept in bed with the usual sanitarium, or modified sanitarium, treatment as instituted here in the hospital.

CLINIC OF DR. CHARLES LOUIS MIX

MERCY HOSPITAL

PRESENTATION OF A CASE OF HOOKWORM DISEASE, WITH SPECIAL REFERENCE TO TREATMENT

January 18, 1916

THIS patient came into the hospital January 10th. His case is interesting, not so much from the point of view of diagnosis as from that of treatment. Still, we must mention a few points of diagnostic importance. The most noticeable thing about him is his *pallor*. As soon as you look at him you notice that his lips and skin are very pale. This drives you at once to an examination of his blood. This showed a total of 3200 white cells, 3,640,000 red cells, and a hemoglobin percentage of 65. Incidentally, his systolic blood-pressure registered 146 mm. of mercury.

Let us first consider the relationship between the number of red corpuscles which he had and the percentage of his hemoglobin; in other words, let us consider his color-index. The color-index is a figure expressing the ratio between the hemoglobin which the blood actually contains and the amount of hemoglobin which it should contain if the corpuscles each contained their normal quota. It is self-evident that if 5,500,000 red corpuscles (the normal number in 1 cu.mm. of blood) contain 100 per cent. of hemoglobin, that 3,640,000 red corpuscles should contain 66 per cent. of hemoglobin. We may, therefore, make the following proportion—5,500,000 : 100 per cent. : : 3,640,000 : x per cent. Multiplying this out and finding the value of x, we discover that 3,640,000 cells should contain 66 per cent. of hemoglobin. We are now in position to determine the color-index. This is done by dividing the percentage of hemoglobin which he actually has,

65 per cent., by the percentage of hemoglobin which he theoretically should have, namely, 66 per cent. Dividing 65 by 66 we get .98 as expressing the ratio, and this we call the color-index.

The color-index is of diagnostic value. Why? Because if it is above unity (1.0) it strongly suggests pernicious anemia; whereas, if it is below unity it suggests a secondary anemia. Evidently, then, this patient, whose color-index is .98, is suffering from a secondary anemia.

We are now ready to give you the results of the differential white count. This also proves to be of great aid in diagnosis. The count shows 23 per cent. small lymphocytes (normal 25 per cent.); 22 per cent. large mononuclears (normal 5 or 6 per cent.); 45 per cent. of polymorphonuclear cells (normal about 65 per cent.); 5 per cent. eosinophil cells (normal 1 per cent.). Such a count is, on the face of it, an evidence of a pathologic condition. The increase in the lymphocytes and the decrease in the polymorphonuclear cells strongly suggest that the changes are not due to a pyogenic organism. The *eosinophilia strongly suggests parasitism.* From the blood-count alone one would say that the eosinophilia, together with the secondary anemia, indicated blood destruction by some parasite. Were the case a primary pernicious anemia his color-index would be above unity. We should also expect to find a few nucleated red cells, an increase of the megalocytes to 40 per cent. or more, a marked poikilocytosis, and no eosinophilia.

The patient is a young man, twenty-one years of age, by occupation a station agent. He was born and brought up near Tampa, Florida, in the sandy zone which is the favorite habitat of the hookworm. He has been living in Iowa the last three years. Inquiring into his previous health, we learn that he had malaria five years ago, from which he completely recovered. Of far greater importance is the fact that while he ran barefooted in Florida he was the victim of a condition known as "ground itch." The last fact is of extreme importance, because almost all cases of hookworm disease in the South occur in individuals who have had ground itch. Indeed, it is thought probable that the hookworm gets into the body from the soil by way of cracks in the

feet. In some localities in the South, where "dirt eaters" live, it is not unlikely that the disease is acquired directly from the soil, the ova passing immediately into the alimentary canal.

Aside from the history of malaria and ground itch there are no other previous illnesses. The patient came to the hospital because he was pale and sleepy, and pallor and sleepiness are his only complaints. On entrance a Wassermann examination was made and proved negative. Because of the eosinophilia the feces were at once examined and showed the ova of the uncinaria. The feces have been examined daily since his entrance and not once have we failed to find the ova. Examination of the blood was made, and the results have just been communicated to you. These merely show a secondary anemia. You will remember that a long while ago, during the building of the St. Gothard Tunnel in Switzerland, a number of cases of an unusual kind of anemia turned up among the workmen. It was at first thought that these were cases of pernicious anemia until pernicious anemia became better differentiated from other anemias. Then it was found that the cause of this particular group of cases was the ankylostoma.

Treatment.—The practice in the Canal Zone in Panama is to make the hemoglobin estimate before starting the treatment. The diagnosis is made by finding the ova of the uncinaria and the hookworms. It is not difficult to make the diagnosis, because it is usually suggested by the blood-picture of anemia, by the sleepiness, and the asthenia of the patient.

There are four things that have been used from time to time in the treatment of this disease, and they have been worked out very accurately as far as therapeutic results are concerned. The first thing was oil of eucalyptus, after that beta-naphthol, next thymol, and, lastly, oil of chenopodium. In 1457 cases, carefully selected with a view of determining the efficiency of these four drugs, it was found that the oil of eucalyptus had an efficiency of 38; the beta-naphthol an efficiency of 68; the thymol an efficiency of 83; and the oil of chenopodium an efficiency of 91. This efficiency may be roughly determined by counting the number of worms in the first stool after the administration of the remedy.

The dosage is different—that of thymol is 60 grains; that of chenopodium 48 minims. Three doses of 20 grains of thymol are given, or two capsules of 8 minims each of the chenopodium for three doses. The routine method is that the patient is given no supper, and is ordinarily given on retiring an ounce or two of sulphate of magnesium. Then early in the morning, in the Canal Zone at 4, 5, and 6 A. M., he is given one or the other of these two drugs, thymol or oil of chenopodium. He is given three 20-grain doses of thymol in capsules, or two capsules of 8 minims each of the chenopodium in three doses at 4, 5, and 6 o'clock. This is followed by one or perhaps two ounces of castor oil at 9 A. M. This is followed by a stool, and all the worms found in the first stool are counted. It has been found that more worms are brought down by the oil of chenopodium than by thymol.

Experience in the Canal Zone is to the effect that thymol cannot be given every day, because it produces too much gastric disturbance. Often there will be a great deal of vomiting and other severe derangements, so it is customary either to give the treatment with thymol at intervals of four to five days and the treatment with the chenopodium every three or four days. It usually takes about five or six series of treatments to clean a case up; it rarely happens that it can be done in one treatment.

How shall you know that a patient has been cured? The first stool after the first treatment is washed through cheesecloth and the worms counted; they may run 60 or 70 or even up to 2000 worms in one stool. During the next few days the stools are watched carefully for ova; they may be found or they may be negative. Then a second course of treatment is given, and again the stool is washed through the cheesecloth and the worms counted. If no worms or ova are found no further treatment may be necessary, but if any are found another course of treatment must be given. The thymol or chenopodium must be continued until there is absence of worms and ova for at least six or seven days, and then you may be sure your case is cured.

We ought to follow this line of treatment, which has been so carefully worked out in the Ancona Hospital with great faith-

fulness. We have no great experience with this disease in the North, but there they have hundreds of cases. In the *Journal of the American Medical Association* there is an article by Bishop and others of the Canal Zone in which they report the testing out of the thymol and chenopodium and they conclude that the chenopodium is much more efficient. Even after failing to drive out more worms by the thymol treatment they got as many as 20 with the chenopodium.

The worms are usually found in two localities—near the lower cecal valve and near the duodenum. It is, of course, necessary to dislodge them from both places, and it may be necessary to give quite a series of treatments to accomplish this.

It used to be the custom in the Canal Zone in giving the castor oil after the thymol or chenopodium to give 50 minims of chloroform by mouth, it being thought that this had a deadening effect on the hookworms and made them easier to dislodge, but it has been found unnecessary to do this with the oil of chenopodium. So we have for an individual treatment the preliminary sulphate of magnesium, followed by thymol or chenopodium, and a final dose of castor oil for the purpose of expelling the dead worms.

The anemia is purely secondary, and as soon as the worms are gotten rid of the hemoglobin begins to come up by itself. It is surprising how rapidly it rises; in a month or two, without any treatment whatever, the hemoglobin often rises 15 or 20 per cent.

I showed you the case this morning not so much for the diagnosis as for the opportunity of speaking a word or two about the treatment of this condition. If you scatter your thymol, giving it over a period of several days' time, you throw your ammunition in part way. The principle is the same as in getting rid of a tapeworm—you must get the remedy through in one large mass. The advantage of the oil of chenopodium is that it is more effective, less toxic, and can be given every three days as against every five days with the oil of thymol. We hope to get this young man well while he is here, although he may have to be here some little time. There is no soreness or tenderness in the abdomen and the anemia is the most striking thing about him.

Of course, in our Southern states the Ankylostoma is very

common. It is interesting to note the way in which the treatment has been shifted, first from the oil of eucalyptus to the beta-naphthol, then to the thymol, and lastly to the chenopodium. Whether we shall subsequently find anything more effective than the latter is hard to say.

Note March 24th.—The patient remained in the hospital for a total of six weeks. In all he took three series of thymol before the oil of chenopodium could be obtained, and three series of the oil of chenopodium at weekly intervals. After the fifth series the ova disappeared from his feces and no worms were obtained. Another treatment was given to him a week later and again no ova were found. It was, therefore, felt that the patient had made a complete recovery and that his parasites had all been killed off.

Meanwhile the patient's physical condition improved markedly. He gained several pounds in weight, his color came back, he soon left his bed and was up and about the ward all the time and was quite active. His cheeks became pink, his ears began to show signs of blood, and his fingernails once more became rosy. When he left the hospital he appeared in every way to be a normal young man. His hemoglobin had gone up to 90 per cent. and his red cells to about 5,000,000. He left the hospital about the first of March.

HYPERNEPHROMA OF THE LEFT KIDNEY: SPECIAL CONSIDERATION OF HEMATURIA

January 25, 1916

DR. MIX: This patient consented to come down this morning for our benefit. (To Patient): What is your age?

PATIENT: Sixty.

DR. MIX: How long have you been sick?

PATIENT: Two or three years.

DR. MIX: How did it begin?

PATIENT: I was ailing in the stomach all the time.

DR. MIX: What did you feel in the stomach?

PATIENT: Pain after my bowels moved.

DR. MIX: Show me where the pain was. (Patient indicates.) It was across the region of the abdomen just above the level of the umbilicus. Was this only after a movement of the bowels or did it come on after you ate?

PATIENT: Both.

DR. MIX: Was it as though there was something griping in there?

PATIENT: No.

DR. MIX: Was it sore when you pressed on your abdomen before the last two months?

PATIENT: No.

DR. MIX: Was the pain brought on when you made water?

PATIENT: No.

DR. MIX: Was the pain ever associated with vomiting?

PATIENT: Yes.

DR. MIX: When did you first vomit?

PATIENT: Two or three months ago.

DR. MIX: That is, then, for the first two or three years of your illness you did not vomit, but during the last two or three months you have been vomiting. What came up?

PATIENT: Foam.

DR. MIX: Did food come up?

PATIENT: No.

DR. MIX: Did it have bile in it?

PATIENT: No, it was white.

DR. MIX: Here one may make this point—there are two types of reflex vomiting which are quite different from each other. One is the reflex vomiting from disease of the upper portion of the abdomen, such as inflammation of the peritoneum about the stomach, pylorus, gall-bladder, or duodenum. Any infection in this locality is capable of causing a reflex anastalsis which sweeps bile into the stomach. There it acts as an emetic and vomiting takes place.

There is another kind of reflex vomiting which is due to disturbances from below. Characteristic of this is the vomiting of pregnancy, the vomiting which takes place in nephritis, or in wandering or floating kidney, in neoplasms of the kidney, or after renal operations; and in this case we have the vomiting of just food or foam and no bile. This patient's vomiting indicates a reflex of another type from the vomiting one would get from a disturbance in the upper part of the abdominal cavity due to a duodenal ulcer associated with peritonitis, or a chronic cholecystitis, or a pancreatitis.

Thus far in this case we have a story of a disagreeable abdominal sensation, which was associated with some soreness on pressure, which was made worse by a bowel movement or the taking of food and, furthermore, a pain which was not colicky but which was due to a soreness and was worse on pressure. Now there has been added to this symptomatology, which up to this point is rather vague and difficult of analysis, another symptom. Since the onset of vomiting two or three months ago there has been a loss of weight of 10 pounds. Whether that loss of weight was due to her vomiting is indicated pretty clearly by this; she vomited only foam, and therefore the loss of weight was not a starvation loss of weight, for she got her food and could have assimilated it. There has been no fever at any time, so it probably was not loss of weight due to infection. It might, of course, be a metabolic or a cachectic loss.

DR. MIX (to Patient): What was it that attracted your attention and made you come to the hospital?

PATIENT: I saw blood in my water.

DR. MIX: One month ago she noticed that there was in her urine the color of blood. In other words, there had been added to her otherwise vague and uncertain symptomatology something that was very spectacular. This bleeding kept up until finally she came to the hospital. She has been here now for eight days and during five of those days she has had no bleeding. When she came in, however, the bleeding was very, very marked. Such a history of hematuria coming on after such a long prodromal history, when correlated with the location of her pain on pressure and her loss of weight, almost gives the diagnosis away at the very beginning.

The ordinary causes of *hematuria* are not very numerous. The unusual and rare causes are fairly numerous. The most common causes are: (1) acute nephritis and acute hemorrhages in chronic nephritis, (2) stone in the kidney or bladder; (3) tumors of the kidney or bladder; and (4) tuberculosis of the kidney or bladder. These four causes account for practically 90 per cent. of the cases of hematuria that one encounters. There are also, of course, occasional red blood-cells found in the urine in conditions of urethral and prostatic trouble, in renal infarcts, in certain parasitic diseases, in so-called essential hematuria, in hemophiliacs, and in severe passive congestion of the kidneys, but in ordinary cases where we encounter hematuria we expect to find one of the four causes mentioned. As a working basis it is not necessary to remember a large number of very rare conditions which may give rise to an occasional red blood-cell. Indeed, in diagnosis, the important thing to know is the usual. The unusual and the occult deservedly take a minor position in practical work.

In a given case we have immediately to concern ourselves with the question as to which one of these four groups of conditions is present. All four of them are very definitely separated from each other by their symptomatology, although there are cases where their symptomatologies seem to blend before careful analysis has been made.

In acute nephritis hemorrhage always takes place. You sometimes find so much blood present that the urine becomes smoky, and even quite reddish at times from severe hemorrhages, such cases being described as cases of acute hemorrhagic nephritis. These acute cases are likely to occur in the acute infectious diseases of children, especially as complications in cases of the acute exanthemata. They also take place in adults from exposure. I remember the case of a man who fell into some icy water who had a very severe attack of acute hemorrhagic nephritis as a result.

The subacute cases bleed very little. The chronic interstitial nephritis patient does not show red blood-cells unless there is added to the picture a secondary passive congestion of the kidney which may produce bleeding. The same is true of chronic glomerular nephritis. Chronic types of nephritis, however, very frequently show red blood-cells in the urine, but very rarely to the extent of producing a red tinge.

In the present case acute or subacute nephritis, or an acute hemorrhage in a chronic nephritis, is excluded at once by examination of the urine and by the history. It is perfectly evident that this patient cannot have an acute nephritis, because she has been ill for a long period, has lost no weight, has shown no edema, has had no uremic symptoms, such as headache or spasms with or without loss of consciousness. Moreover, the urinalysis shows the absence of casts and an unchanged specific gravity. Furthermore, her cardiovascular apparatus shows none of the signs ordinarily associated with a nephritis, either acute or chronic.

For the same reasons that an acute nephritis is not present, an exacerbation of a chronic nephritis cannot be the explanation of the hemorrhage. Were there a chronic nephritis present we should expect at her age a high blood-pressure with numerous arterial changes, with a very much enlarged left ventricle of the heart and definite urinary findings in the way of casts, especially of the granular variety. We would also expect to find running through her history some sign of renal intoxication, which in her case we do not find. For these reasons a diagnosis of either

an acute nephritis or of an acute hemorrhage in a chronic nephritis does not seem tenable.

We have next to consider the question of the presence of stone either in the pelvis of the kidney or in the bladder. In each instance hemorrhage is apt to occur. If the stone be in the kidney it may or may not be associated with a large amount of pus. There is one change, however, which is almost always noted in the red blood-cells themselves in cases of renal hematuria from stone; that is, the individual red cells are washed out in appearance. They belong to the so-called "shadow cells," consisting merely of rings with little or no hemoglobin. The hemoglobin, as a rule, has already been laked out into the urine.

Moreover, you can tell kidney blood from bladder blood, as a rule, by *fragmentation* of the red blood-cell. If the cells show no fragmentation, if they are not fragmented, they probably come from the bladder. If, however, they are fragmented, have lost their hemoglobin, they probably have come from the kidney. Then, again, bleeding from the kidney is usually an oozing and does not produce clots. The blood comes down a little at a time with the urine, and it is rare for clots to form in the bladder from blood from the kidney. So if clots are passed the probabilities are nine out of ten that the blood is from the bladder. This fact affords a very good method of differentiation between blood from the kidney and blood from the bladder.

Moreover, since the blood from the kidney comes down slowly and with the urine mingled with it, you will find that the urine collected in three tubes is uniformly bloody; but if the blood comes from the bladder this will not be the case. In cases of hemorrhage from the bladder caused by stone the blood is apt to be passed at the end of micturition. The urine which is passed first is less apt to be very bloody than that which is passed last. The corpuscles in such a specimen, when examined under the microscope, are found to be fresh, unwashed corpuscles.

But more important than the different types of red blood-cells in the two conditions, because exceptions occur both ways, is the subjective complaint of the patient. The patient with a kidney stone usually has a renal colic, whereas the patient with a bladder

stone has the pain in the bladder itself. Moreover, if the stone is in the kidney it is usually quiescent for long periods of time. Pain comes only when nature attempts to move the stone. As soon as motion ceases the pain ceases. The same condition holds true in the bladder, but to a markedly lesser extent because the stone has a tendency to remain in the bladder rather than to be passed out. In this condition the pain is apt to be more severe when the bladder is empty and contracting upon the stone, and less severe when the bladder is partially filled. Usually a stone in the bladder causes a laceration or excoriation of the bladder wall and leads to very frequent urination, which is not observed in cases of renal stone. Moreover, in cases of renal stone the pus in the urine is apt to occur in a series of paroxysmal outpourings, whereas in bladder cases the pus is usually eliminated at a steady pace.

Taking these factors into consideration, since this patient has had none of the symptoms mentioned, and since her urinary findings do not conform to the findings of stone in the kidney or stone in the bladder, it is evident that these conditions cannot account for her symptomatology. We must look for something else.

Another fruitful cause of red blood-cells in the urine is tuberculosis either of the kidney or the bladder. Tuberculosis of the bladder is seldom primary. It is the kidney which is first involved and which subsequently may lead to involvement of the bladder, but curiously enough, although the process begins primarily as tuberculosis of the kidney, the early symptomatology is confined to the bladder. The first thing that the patient notices is marked bladder irritability with increased micturition. Next the pus begins to come. Hemorrhages are comparatively small in amount and usually quite rare. Indeed, in most of the cases the patient never knows that blood has appeared in the urine. It is only the man who makes the microscopic examination who makes this discovery. These cases usually are associated with a small amount of fever, showing an early afternoon temperature and marked bladder irritation with the presence of pus in the urine.

It is evident, if you recall the symptoms which this patient presented, that none of these conditions hold true. Moreover, to be doubly sure she was given a tuberculin test. It resulted negatively.

Lastly there remains as a cause for hematuria tumors of the bladder and kidney. Of the latter the most common are the sarcomata and hypernephromata. Of the former the most common are the carcinomata and the polyps. In all four of these conditions we do not have the steady appearance of small quantities of microscopic blood, but we have from time to time spectacular hemorrhages in which a great amount of blood is passed. In cases of hematuria from sarcomata and hypernephromata it is most unusual that clots should be passed, whereas in cases of hematuria from the bladder wall or from bladder polyps clots are by no means uncommon. Indeed, clots may remain in the bladder, and it may be necessary for them to be removed surgically in the latter conditions.

In the case of this patient clots have not been passed, although she has had large hemorrhages in which the urine has been very bloody. This absence of clots would seem to indicate that the blood came from the high level rather than the low level. Moreover, in her case, there being no bladder symptoms at all, no increased frequency of urination, no tenesmus, no bladder pain, and no painful micturition, and, furthermore, since she has under the left costal border a mass which lies behind the colon, it is almost self-evident that the blood must come from the left kidney.

Renal neoplasms and stone are usually differentiated from each other by the presence or absence of renal colic, but there are many exceptions. There are many cases of stone where there is no colic, and many cases of neoplasm where there is colic. One can't be dogmatic—one can't say that if there is renal colic the probability is that there is a stone, and if not that there is a neoplasm. One can say, however, that in a neoplasm there is more likely to be a long-continued hemorrhage, and in a stone a hemorrhage which is more likely to be intermittent. So when you see a case like this where there has been blood for almost a month you are pretty nearly certain that you are dealing with a

neoplasm and not with a stone. When you take still further into consideration the loss of weight you are still more convinced that it is a neoplasm.

In the examination of the patient there is found, situated high up, at the level of the umbilicus and $2\frac{1}{2}$ inches to the right of the midline, a tumor mass which is fixed against the abdominal wall behind. It moves very little with respiration, and in this respect behaves as a tumor of the kidney should behave. When we have her raise herself to a sitting position we can feel the kidney and the tumor much more plainly. When we place her on the right side we can with the left hand thrust the kidney toward the anterior abdominal wall from behind and palpate it with the fingers of the right hand in front.

When we felt this large left kidney the inference was that it was a neoplasm, and the next consideration was as to the nature of the neoplasm. The nature of the neoplasm is suggested by two or three important facts. One of them is that her illness has persisted over two or three years. Another is that sarcomata of the kidney are usually pretty rapid-growing tumors. Hypernephromata are frequently slow growing, being at times semimalignant. Furthermore, hypernephromas are very vascular tumors, and they are tumors which are very apt to bleed. So vascular are they that even their metastases have been known to pulsate.

From what has thus far been said it must be quite evident that the cause of the blood in this case is clearly a neoplasm of the left kidney. The question which we have to consider is whether there is any disease of the right kidney and the nature of the neoplasm in the left. In order to be absolutely certain on this point it is necessary that the patient have the two ureters catheterized. This was accordingly done in this patient, and it was found that the blood all came from the left kidney. Moreover, the right kidney, during the test, eliminated more urine than the left one. Perhaps it is not strictly truthful to say that no blood came from the right kidney. Anyone who has had experience in catheterization of the ureters knows that it is not an infrequent occurrence after catheterization to get a small amount of traumatic blood in both catheters. A few red blood-cells appeared in

the urine from the right kidney. This does not indicate that the right kidney is diseased, but that there was a slight amount of trauma from the catheter. From the left kidney, however, a large amount of blood was obtained, the urine from the left kidney being bright red in color. The bleeding, correlated with the existence of an enlarged kidney, is good evidence that we are dealing with some sort of a vascular tumor of the left kidney.

Sarcomas also bleed, but they do not bleed over so long a period of time as a rule, and do not so often occur at the age of sixty as hypernephromas do. We expect to find sarcomas of the kidney in children—not in old people. There is another thing about hypernephromas that is not present in this case, and that is, that they are very prone to have metastases, many of which occur in bone. Hypernephromas are semimalignant; they may last four or five years before producing a fatal termination. They may have metastases, but in many cases they do not. We recently had at this hospital a patient with a hypernephroma of the kidney where there was a metastasis in the arm-pit, one or two on the chest, and one in the cervical region of the neck, and curiously enough this case of hypernephroma—as proved by operation by Dr. E. Wyllys Andrews—never had any blood in the urine. In this particular case at times the whole urine was collected, divided into ten parts, and no blood found in any one of the ten centrifuged tubes. So it was proved that this particular case was not bleeding even in microscopic quantities. The reason we went to this length in our search for blood is because hypernephromas notoriously bleed and this particular case did not do so. The reason they bleed so easily is because of their vascular character. The spread of the metastases is venous, ordinarily from the vena cava, into which the renal vein empties, and from there to other parts of the body. We have searched the surface of this patient's body for signs of metastases, but we find none. We have told her the nature of her trouble, but she does not wish to be operated upon. She thinks she would succumb to the operation and that it is best for her not to have one. She has been further encouraged because the bleeding has stopped for the past five days. Why? Because she has been lying very quietly

in bed. If she were about and on her feet I have no doubt the bleeding would return very quickly.

Is there any treatment for hypernephroma except surgery? Of course, there isn't. Many cases of hypernephroma can be cured by excision of the tumor, and it should be done in this case, but the patient will not submit to it. If operation is refused, it is useless to hope for anything from any other treatment. The *x*-ray has been tried in these cases with some measure of success. In the patient I spoke of as having been recently in the hospital the size of the neoplasm began to decrease, and after a few *x*-ray treatments it was only about half the original size. Curiously, with the reduction of the main tumor, there was a reduction in the size of the metastases, but after the tumor was reduced to one-half its former size nothing more happened. The anemia went on, the red cell-count fell to approximately 2,000,000, and the case terminated fatally.

In many cases of hypernephroma the initial symptoms suggest cardiac degeneration. The patient last mentioned in the beginning had shortness of breath and palpitation of the heart and his heart was distinctly weak. These complaints antedated the finding of the neoplasm in the kidney, which was not found until about a month after the development of his cardiac symptoms. That man ran rather a rapid course; the fatal termination came within two years' time. Many cases extend over five years or more.

Visiting Physician: Do you recommend removal of tumor growths in people under twenty years of age?

Dr. Mix: Yes; this is the way I look upon it: those cases occurring early in life are all going to be fatal, but there is a possibility of saving an occasional one by operation.

Visiting Physician: I thought it often went to the other kidney after operation.

Dr. Mix: I wouldn't say that neoplasms were very prone to invade the opposite kidney; tuberculosis, however, is. I think one may say, as a rule, that tuberculosis will almost always invade the opposite kidney, but that neoplasms are not nearly so likely to do so.

CLINIC OF DR. ISAAC A. ABT

MICHAEL REESE HOSPITAL (SARAH MORRIS MEMORIAL HOSPITAL FOR CHILDREN)

RACHITIS

Frequency and Geographic Distribution. Etiology. The Part Played by Calcium in the Metabolism of Rickets. Symptomatology. Complications. Prophylaxis. Full Management. Treatment—Dietetic, Hygienic, Medicinal. Value of Phosphorus in the Treatment.

Case I.—This little boy whom I present to you this morning is two years old. He was admitted into the Sarah Morris (Children's) Hospital three days ago. He was a full-term baby, born after a long, hard labor, and delivered by forceps. He was considered normal at birth. He was breast fed for three weeks. Then mother's milk was thought insufficient in quantity and the child was given artificial food. The formula of the first food preparation contained 16 ounces of certified milk, 16 ounces of water, and one tablespoonful of cane-sugar. He received this from the time he was six weeks old until he was one year old, so it is reported to us. From the first year to the second year he was given certified milk in the quantity of 1 quart a day. He was brought to the hospital several days ago by the grandmother, who thought that the child was undeveloped in every respect. The grandmother says he does not talk, though our nurses tell us he says some words. He cannot walk, he stands poorly, and the child is restless. The parents attribute his condition to the fact that he refused solid nourishment and partook only of milk. The father says the baby is in good health. The mother is in delicate health and is unable to look after the welfare of the child. She had one miscarriage eight months ago.

The blood examination shows 60 per cent. hemoglobin, 3,600,000 red cells, 7,000 leukocytes, of which 56 per cent. are neutrophils, 35 per cent. mononuclear leukocytes, and 9 per cent. large mononuclear leukocytes.

The urinary examination gave normal results.

The von Pirquet test did not react, and we assume that he is free from tuberculosis.

It was found, too, that the blood gave a 4 plus Wassermann reaction. We may say in passing that a baby who is born with congenital syphilis is very likely during the course of the first year to become rachitic. We do not think that syphilis is the cause of rickets, nor that there is any specific relationship between syphilis and rickets; but on account of the chronic infection and on account of the malnutrition, which is nearly always associated with syphilis, we assume that syphilis interferes with the normal nutritive processes of the body and tends toward the occurrence of rickets.

Before we proceed further with the examination I desire to call attention to the note in the history sheet, which says that the baby will take no nourishment except milk and an occasional cracker. This notation is of more than passing importance, and has a bearing on the nutritive condition of the child. Children who take nothing but milk, particularly after the first year, or after eight or ten teeth have erupted, who forego all other foods, such as vegetables, cereals, broths, fruit juices, and so on, almost invariably become anemic. The same is true of breast-fed children; those who are kept for a long time on an exclusive breast-milk diet also tend to become anemic.

I have already called your attention to the fact that the iron content of milk, whether it be from the human being or from the cow, is deficient for the needs of the child. There is less iron in breast milk than in cows' milk. The Czerny school has suggested the hypothesis that the baby comes into the world with iron stored in the liver or in other organs, so that when the food, namely, the milk, does not offer sufficient iron for the needs of the body, this reserve quantity may be called upon to supply the deficiency.

This baby has no increased temperature; the highest recorded temperature is 99° F. The pulse varies considerably and is inclined to be somewhat rapid, though you know we have agreed that the normal pulse-rate of the child is best ascertained when he is quiet or asleep. You will note that this baby's head is large, and when one lifts it, it gives the impression of being heavy and massive. The fontanel is still open, though it is depressed. The head is broad from side to side and has a square appearance, especially in the frontal region. You will notice the protuberance over the frontal eminences; some protuberances are noted over the parietal bone. The clavicles are rather acutely bent, so that each seems to form an apex about half-way between the sternal and acromial juncture and slopes toward either side.

On examining the bony thorax, one notices the prominences on the junction of the bony with the cartilaginous portions of the ribs, and these prominences or bead-like enlargements constitute the so-called rachitic rosary. Note, too, a horizontal depression over the lower right half of the thorax. This corresponds with about the point where the diaphragm is inserted and is slightly above the upper margin of the liver. This depression continues to the left side of the thorax to about the same level and is spoken of as Harrison's groove. Otherwise there are no marked deformities of the bony thorax. We note, too, that the localization of the rachitic process varies with patients. Sometimes rachitis is most marked in the bones of the skull, and at other times the disease preponderates in the bony thorax or again in the spine, though it may be general and widely distributed, so that all bony structures are affected.

In this case that we are presenting to you the head seems to be more definitely rachitic than the thorax; the changes in the thorax are of moderate degree.

If we examine his upper extremities we note that there is a slight enlargement of the epiphysis at the wrists. The forearms are slightly bent. The bones of the upper extremities are of normal length.

In passing, let us note his musculature, which is very much relaxed. When we speak of the muscles as being flabby, they

lack tonicity. Not only is this so of the muscles of the upper extremities but also of those of the chest and back. Note the pectoralis major, for instance; it lacks tone, is soft and flabby, and seems to be very poorly developed.

Let me call your attention to his teeth: For a boy two years old, they seem of poor quality; many of them contain cavities, some are brittle and decayed. Some of the lymph-nodes are enlarged in various locations. Enlarged lymph-nodes may be felt in the neck and in the axilla. His abdomen is not protuberant; the musculature is relaxed and atonic; the abdomen seems large, though there is no tympany and no abdominal distention.

We now turn to the lower extremities. You will note that if the knees are brought together the legs make quite a sharp angle in the opposite direction; that is, there is an outward deviation of the legs when the knees are approximated. The condition is then one of knock-knee or genu valgum.

If we examine the femur on either side you will notice that it is curved, the curvature is very marked, and that it is forward in direction. These curvatures of the femur may occur in many directions and may be very difficult to describe. If he is permitted to stand, you will note that his feet are very flat; he stands more on the inner side of his feet and the arch is not preserved. If he is placed in a sitting position, you note that he is unable to support his back and he leans forward; owing to the weakness of muscles and spine he is unable to sit erect. Even his gluteal muscles seem weak; the skin wrinkles, and they seem insufficiently contracted to permit him to sit erect. His spine shows no marked deviation.

In young children with rickets a systolic murmur is frequently heard over the fontanel. This was first described by Dr. Fisher, a Boston physician, and the murmur was thought to be due to a narrowness of the carotid canal. This is frequently heard in anemic infants. This murmur cannot be heard in this child.

The liver is slightly enlarged; it may be felt two finger breadths below the costal arch, but it cannot be felt to the left of

the median line. The liver is not particularly hard, and this slight increase in size has no pathologic significance.

The spleen is not palpable.

Percussion of the abdomen gives the usual and normal findings, and no additional information is obtained by palpating the abdomen. The umbilical ring in this case is closed. The recti are pretty well approximated. There is no hernia. The testes have descended normally. There is no phimosis.

Summarizing, then, we have here a rachitic child, two years of age, who shows changes in his skeletal structure and is very much retarded physically, particularly in his static conditions. He cannot walk; stands only when supported and sits with difficulty. He seems of normal intelligence. He eats poorly; must be urged, sometimes forced. He sleeps well; seems to be happy and contented, particularly when he is not being examined. There are no definite syphilitc manifestations, though the enlarged lymph-nodes and the rapid decay of teeth may bear some relation to the syphilis which we believe was demonstrated by a very striking active blood reaction.

Case II.—I desire to present another child suffering from the same disease. This little girl, three years old, born at full term, spontaneous delivery, cephalic presentation, was thought to be a normal baby at birth. For the first seventeen months of her life she was breast fed, then she was put upon a soft diet—that is, soup, milk, bread, cereals, and some fruit. The first tooth appeared at ten months; she began to talk at fifteen months; she cannot walk at the present time, though she is three years old. Indeed, she was brought to the hospital because she was unable to walk. She is small for her age, has a very poor appetite, and desires to eat only milk and potatotes. Her mother says she can stand when supported. Both mother and father report themselves in normal health. Mother had one miscarriage.

The blood-count examination shows 85 per cent. of hemoglobin, 14,000 leukocytes, of which 63 are neutrophils, 14 small mononuclear leukocytes, 20 large mononuclear leukocytes, and 3 transitional cells.

The Wassermann examination was negative.

Now, examining this child, you will note she is small for three years of age. The muscles are everywhere flabby or atonic. The brows are slightly overhanging. The frontal bones present bosses or protuberances. The fontanel is still open at three years of age, though the sutures are closed. The parietal bones also present bosses on both sides. The thorax shows some slight prominences anteriorly over the sternum. The baby has a rosary. There are lateral depressions in the axillary lines. The lower ribs flare. The spine shows a slight curvature toward the left. The epiphyses are enlarged at the wrists. The child has enlarged glands in the axilla and at the angles of the jaw. The spleen is not definitely palpable. There is no very marked curvature of the long bones. Note, too, that the child has not erupted the full number of temporary teeth; dentition is extremely retarded.

Let us consider this disease—rachitis—in more detail. The disease was first described accurately and in detail by Glisson, an English clinician, about 1650. Glisson's description is very accurate, his observations were keen, and while much has been contributed during the many years which have elapsed between the time of Glisson and now, nevertheless, very little has been added which would much enrich his clinical descriptions.

Geographic Distribution.—Rachitis occurs widely distributed over the entire earth, though in certain regions where people live in a natural state, that is, out-of-doors, the disease is not so common as in overcrowded and overpopulated districts. Rachitis occurs in the country as well as in the city, though the city contributes the largest proportion, and particularly those quarters where overcrowding and unsanitary conditions prevail. It has been said that in the city of Berlin 90 to 98 per cent. of all the children who are examined show some evidence of rickets. In the northern lands, as in Greenland, rickets is said to occur very seldom. Rickets is said to occur seldom in Japan. Rickets is also thought to be uncommon in the tropics and the subtropical regions; it is not of frequent occurrence along the southern Italian shores, though in this country, as everybody knows who visits the dispensaries and hospitals of our large cities, rachitis

among the Italians is of very frequent occurrence. The same thing is true among colored children. Rickets occurs so frequently among these children that the absence of rachitic changes is the exception and its presence is the rule. I have often thought that these people who have been removed from their natural environment, that is, from their Southern homes and their out-of-door life, from their location near the equator, and brought into a colder and more rigorous climate, were more prone to rickets than those whose forebears had for generations been accustomed to the rigorous north temperate climate. But the disease occurs among all classes; it is found in the homes of the well-to-do more often than is generally supposed.

The **etiology** of rachitis is an extremely difficult chapter. Every possible theory has been invoked and, after having been subjected to clinical tests, experience, and time, has been rejected. While we know that more children become rachitic on artificial food than on breast feeding, on the other hand, we know that a certain proportion of breast-fed children may become rachitic. In any event, it has been impossible to point out the particular element in the diet, if there be such, which is the underlying cause in the production of rachitis.

The heredity of rickets has received much attention. If rickets be due to hereditary influences, then one would expect that the infant should be born with rickets or show rachitic changes very shortly after birth. Congenital rickets is of extremely infrequent occurrence. One cannot deny, however, that in certain families there is a disposition to the occurrence of rickets. We know, for example, that in certain families all the children at a very early age show manifestations of rickets. In these cases, too, it usually may be shown that the mother or father, or both, still show some evidence of rickets. An interesting case is cited in the literature. A certain woman contracted several marriages; the children by her first husband, who himself had suffered from rickets during his infancy, were all markedly rachitic; the children by her second husband were also rachitic; and in the third event, the father, who was a robust man, had no evidence of rickets, the offspring in turn remained remarkably free from

the disease. The story would prove that under certain conditions rickets is hereditary, depending upon the previous existence of the disease in one or both parents. While this isolated case and many others may be cited in favor of the view that rachitis may be hereditary, nevertheless we must say that the influence of heredity in rickets cannot be considered definitely proved.

Tuberculosis and syphilis contribute to the occurrence of rickets; not that tuberculosis and syphilis are etiologic factors in the narrow sense, but babies who are born syphilitic or those who acquire tuberculosis are likely to suffer in the general nutritive processes, and as a result rickets occurs secondarily. In other words, syphilis and tuberculosis are predisposing factors to the production of the disease. Children suffering from whooping-cough, measles and its complications, and prolonged anemia are likely to become rachitic. No one kind of food can be held responsible. It is not true, as has been supposed, that food deficient in fat is the cause of this condition, nor is an excess of carbohydrates or an excess of protein the cause. In the same measure, it may be assumed that overfeeding of fat in itself is not a cause. Any and all foods which lead to protracted disturbance of digestion, or disturbances of the metabolic function, particularly so far as the appropriation of calcium is concerned, are likely to lead to rachitic changes in the bones and consequent disturbances of nutrition.

It is usually stated that rickets has its seasonal occurrence; that it is more likely to occur in the spring of the year than any other time. It has been suggested that the reason for this increased prevalence of the disease at this time of the year is due to the fact that children remain indoors the greater part of the winter; they frequently live in unsanitary, poorly ventilated homes during the entire winter; that the infants suffer from want of fresh air and proper ventilation as well as sunlight. The homes are usually overheated, though there is evidence to show that even in the better ventilated homes and among the well-to-do classes rachitis occurs.

Pathogenesis.—For many years experimental work has been conducted to ascertain the etiologic factors in the production of

rickets. Feeding experiments upon animals with the various elements of food have been recorded in the literature of thirty years ago. In the most recent times the experimentation has been restricted more to the effects of calcium metabolism. The questions propounded have been, "Does rickets result from giving too little calcium in the food or is the normal calcium content of the

Fig. 223.—Italian child, two and one-half years old, showing rachitic changes in head, thorax, abdomen, and extremities.

food not absorbed in sufficient amounts, or is it absorbed, and are the bones not able to assimilate it in the normal manner?" The results which have been obtained are not altogether uniform. If animals were fed on a calcium-poor food or an acid food, or a combination of the two, a disease condition occurred which resembled rickets very markedly.

Much investigation has been carried on in an attempt to prove that rickets is due to a perversion of the internal secretions. Many workers have been led to study the relation of internal glandular secretion to rickets; thus, Stöltzner, in an elaborate investigation, thought that the disease was due to a disturbed function of the suprarenal glands. Subsequent research in clinic

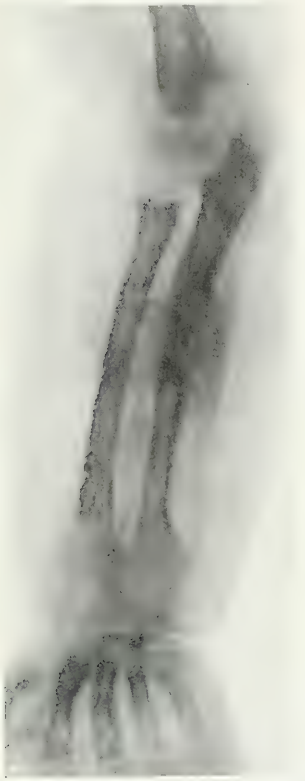

Fig. 224.—Florid rickets. Slight differentiation between bone and cartilage owing to the lack of calcium. Fracture of ulna between upper and middle third. Callous is deficient in calcium.

Fig. 225.—Knee-joint of eleven months' rachitic child, showing reparative changes indicated by the deposit of cartilage in the osteogenic zone.

and laboratory has failed to corroborate this view. Similarly, perversions in the function of the thyroid and the thymus have been invoked to explain this disease, but the evidence has been altogether insufficient to explain the etiology. The parathyroid has been more recently subjected to experimental study. Ani-

mal investigation points to the fact that this glandular structure has some important function in calcium metabolism, possibly in combining the calcium, thus leading to its retention in the body. When the gland is excised, marked phenomena, such as tetany and convulsions, occur with striking regularity, though there is no conclusive proof that the parathyroid or its secretion plays a constant etiologic rôle in the production of rickets.

If we should attempt to prove that rachitis is caused by a primary deficit of calcium, then we might administer to animals food which is low in calcium to observe the effect experimentally. This has been done, and the results have shown that changes occurred which were similar to those in rickets, though when the bones were subjected to more careful chemical and microscopic analysis it was discovered that the disease was not true rickets. This artificially produced disease has been designated as pseudorickets or, more properly, pseudorachitic osteoporosis. This condition represents the physiologic reaction of an organism with healthy bone to a calcium-poor food. The bone substance is qualitatively normal and quantitatively deficient in amount. Therefore a deficit of calcium in the food cannot be the cause of rickets.

The second point in consideration of the calcium factor leads to the following question: Granting that calcium is present in the food in sufficient quantity, does some failure occur in its absorption, or is it too rapidly eliminated before absoprtion is possible? In some cases it is conceivable that the calcium is ingested in normal quantity, but is lost to the body on account of some defect of digestive function, or that it finds its way beyond the digestive tract, is brought to the bony tissues, and for some reason is not utilized by the bone and consequently must be excreted by the organs of elimination. Metabolism experiments seem to warrant the belief that calcium is not absorbed or utilized by the bone because of its failure to be retained in the body. This may be due to a defect in digestion or to an inability of bone cells to combine in normal manner the calcium salts, as described above. It has been shown by observers that in some instances more calcium was eliminated from the body than was ingested, and these authors

think that milk-fat has an unfavorable influence on the retention of both calcium and magnesium. They believe that on account of an excess of fatty acids in the intestine, there is a combination of the fatty acids with the calcium and magnesium salts of the body, which leads to the formation of soaps, which are eliminated as such. Birk, who has been carrying on this investigation, believes consequently that rickets is due to a primary disturbance of fat digestion. This hypothesis is not on a firm foundation, though, if it could be ultimately proved that rickets depends on some form of indigestion, such an explanation would reconcile clinical data with scientific investigation, and would clear up many gaps in the knowledge of the disease.

Rachitis occurs in children who are fed on many different kinds of foods, even breast milk. In nearly every case of rachitis, irrespective of the kind of food, some evidence of indigestion is present for a prolonged time.

The following hypothesis of Stöltzner seems plausible and has been widely accepted as to the etiology of rickets, namely, that the newly formed bone remains uncalcified because it is not in condition to be impregnated with the calcium which is brought to it in the body fluids. When the calcium is rejected by the bone it must be excreted, and it returns to the intestine, where it combines with the fatty acids to form soaps. If this is true, it is necessary to believe that the formation of soaps is due to excessive elimination of the calcium instead of being the result of an abnormal fat digestion.

Pathology.—A fresh rachitic bone bends easily, is frequently cylindric in shape, and is lacking in normal consistency. If the periosteum be examined it is noted that it is thick and red as a result of hyperemia. Such a bone is consequently swollen, thickened, softened, and subject to fractures. The amount of mineral matter in the bone is considerably reduced. If section be made of the long bone, it is found that there is an increase in the dimensions in every direction. The walls of the diaphysis are thickened, the wall of the medullary canal is narrowed, and the marrow is congested. At the point where the bone and the cartilage join, the cartilage shows considerable thickening exter-

nally, and it appears to be driven forward. The zone of cartilaginous growth is darker, swollen, and plainly increased in width. The limiting zone between the bone and the cartilage, which is normally straight, tends to be wavy or irregular.

The rachitic changes in bones are characterized by disturbances of the endochondral as well as periosteal ossification. It will be remembered that the growth of bone in its long axis occurs as follows: From the epiphyseal margin—that is, so-called

Fig. 226.—Severe florid rickets in a two-year-old child. Broadening of ends of diaphysis.

proliferation zone—columns of cartilage cells form. As a result of the deposition of calcium salts in the cartilaginous matrix a provisional calcification takes place. As the vessels develop and absorption of matrix takes place, provisional marrow spaces are formed. In these spaces, due to the activity of the osteoblasts, genuine bone-tissue is formed. The proliferation zone in normal bone is small and runs in the long axis of the bone. The growth in thickness takes place by the deposit of lime salts as the result of the periosteal growth. At the same time in the growing bone

processes of resorption, together with deposits of calcium substance which precede the actual bone formation, takes place. Now, if rickets is present in developing bone the columns of cartilaginous cells become increased in length; the zone of growing

Fig. 227.—Leg of two and one half year old child. Rickets in process of healing. Marked curvature of fibula infantum of middle portion. Fracture of tibia in upper third of the shaft.

cartilage is broadened and is extraordinarily filled with blood-vessels.

The provisional calcification is inhibited and occurs only in small foci on account of the abnormal vascularity of the marrow spaces; absorption occurs and the calcification leading to bone formation occurs very slowly. Now, as a result of these disturbances, the characteristic conditions for rickets are brought

about. The proliferation zone is increased in breadth and the edges of the zone of calcification are irregular and broken. The rachitic disturbance in the growth of bone consists of hyper-

Fig. 228.—Severe rickets in a child of two years, with marked curvature of bones of thigh and leg.

emia, excessive production and persistence of the osteoid tissue, and, on the other hand, delayed calcification. As a result of the rachitic processes, the long bones become thicker and plumper and markedly distended or enlarged in the region of the

epiphysis, and during the florid stage the bone is softer and more elastic and easily bent. The result of this is that moderate traumas, even the simple pulling of the muscle, the effort of sitting and standing, lead to curvatures, bending, infractions, and even to severe deformities. On the squamous bones one may observe on account of increased resorption abnormal thinness, or, on the other hand, on account of overproduction of osteoid tissue, thickening and swelling. Another change occurs during the healing process of rickets. The overproduction of osteoid tissue becomes calcified and results in thickened, curved, or bent bony tissue.

Symptoms and Clinical Course.—While the most characteristic symptoms are those on the part of the skeletal structures, there are some general symptoms of importance, which may precede by several weeks the changes which are observed in the bones. These symptoms are manifested by restlessness. The children do not sleep well, cry and whine, and are very sensitive to pressure over bony structures; children bore their heads in the pillows, rotate their heads, and literally wear the hair off from the occiput. Sometimes the children lie on one side, or in other cases they prefer to lie on the face. If they are carried about, the head appears heavy, and the forehead falls upon the shoulder of the one who is carrying them. These children perspire profusely, particularly about the head; the perspiration is increased during sleep and during the attacks of crying. In the morning the pillow is wet from the perspiration which has ensued. The perspiration is often of a sticky consistency and smells sour and reacts acid. As a result of the perspiration sudamina and eczema may occur. The vasomotor irritability of the skin is increased, so that wherever the child is touched red spots appear. These children very soon lose the normal color of their skin and become pale. These children learn to walk late; they lose this ability if they are seized with rickets after the walking has begun. As a rule the general symptoms precede by two or three weeks the definite changes in the skeletal structures.

The Manifestations on the Part of the Skeletal System.—The changes produced by rickets on bones consist of softening and deformity. The softening occurs principally in the flat bones and

in the diaphysis of the long bones. Deformities may occur in any bone as a result of rachitic processes. The epiphysis of the long bones and the junction of the cartilaginous with the bony portions of the ribs become swollen, and in the epiphysis the swelling becomes marked and causes the diaphysis to appear small or thin, whereas the diaphysis itself may have become thickened on account of the rachitic processes. On the joints of the hands, feet, and knees the epiphysis may be increased to double the normal size. Moderate deformity of the long bones is frequent, though a pronounced curvature usually depends upon more or less severe trauma. Indeed, the trauma may be so severe as to produce fracture. Infractions which occur in rickets are not infrequently overlooked and are thought to be more or less severe curvatures. The force of the trauma may be of very mild degree, and, particularly if the bones are very much softened, sometimes changing the child in bed is sufficient to produce the trauma which causes fracture. The clavicle, the ribs, bones of the forearm, and femur not infrequently suffer from these infractions. Forcible muscle contraction may lead to the same results or, indeed, of somewhat less frequent occurrence are the fractures produced by convulsive seizures. Protracted rickets may interfere with the growth in the long axis of the body. It should be remembered, too, that curvatures produced by rickets may cause an apparent diminution in height. It is also true that after the florid stage of rickets has passed there may be a period of arrested growth in the long axis. Indeed, it is a fact that during the florid stage of the disease the bones may not develop normally in growth. Those individuals who later in life are proved to be small in stature on account of rickets were probably affected with the disease somewhat later in their childhood. The static changes which occur in rickets are closely related to the disturbances of motor function. It is true, though, that some children learn to walk at the usual time in spite of the rickets. In the majority of instances, however, rachitic children learn to stand and to walk months later than those not so affected. It has already been noted that children who have already learned to walk and are overtaken by rickets lose this ability. The disturb-

ance of motor function of the extremities is often combined with a marked relaxation and an increased passive motion of the joints. This is particularly noticeable in the ease with which the joints of the hands and feet may be moved in any direction in rachitic children. In severe cases the relaxation is so great that one is reminded of a pseudoparalysis. The cause of this, to a

Fig. 229.—Severe rickets in a child of two years, with marked curvature of bones of upper extremity.

certain extent, lies in the fact that muscular action in florid rickets produces pain, and this in turn tends to inaction of the muscles. Rachitic children who have learned to walk show in many cases some deformity of the pelvis and the lower extremities and, in addition, an unsteady gait and a tendency toward fatigue.

The *skull of rachitic children* is usually somewhat larger than in normal children of the same age, and this often leads to a mis-

conception that hydrocephalus is present. It is true that in some cases hydrocephalus may be present, but, as a rule, the dis-

Fig. 230.—Severe rickets in a child of three years. Extreme curvatures of bones of thigh and leg associated with fractures.

proportion between face and skull depends upon the fact that the face is abnormally small and that the head is somewhat enlarged, due to the rickets. The growth of the face bones is

retarded. The upper jaw is narrowed in its transverse diameter, though it may be prolonged, and may project in its anterior portion. The lower jaw is diminished in size in its anteroposterior direction. The ramus of the jaw is short and plump. The inner margins of the teeth of the upper jaw often strike the external margins of the molar teeth of the lower jaw. The incisors of the lower jaw are sometimes crooked and point inward toward the cavity of the mouth. Indeed, rickets tends to cause irregularities in position, growth, and structure of the teeth. In addition, too, rickets tends to delay dentition. The normal intervals of eruption of the teeth are disturbed, and very frequently the temporary dentition is not complete until the third year of childhood, whereas it should have been complete at two or two and a half years. Possibly more characteristic than any other factor connected with dentition is the observation that in rickets the teeth do not erupt in their normal turn and without any regard to their symmetric arrangement; for example, a child may have erupted his teeth out of the usual order, and then a long pause occurs before another group of teeth appear, instead of the expected group appearing as we are accustomed to find them in normal babies. It has been maintained that considerable difficulty sometimes ensues in the eruption of teeth in rachitic children. This much-discussed point need not call for prolonged discussion. The pathology of teething is of ancient origin and has been discussed by philosophers and physicians of all times. The question still remains to be settled.

Fig. 231.—Child of two years. Forearm shows rachitic changes in shaft and epiphysis with evidence of reparative process in diaphysis and epiphysis.

Whether the structure of the teeth themselves is affected by rickets—that is, whether the teeth of rachitic children tend to decay early and to fall out—is not definitely settled. It is known that many rachitic children have teeth which are of good quality and tend to be free from decay for the usual length of time. It

Fig. 232.—Rickets in a child of fifteen months, showing bony changes and infraction.

has been assumed that in those cases where the teeth decay and fall out early, hereditary syphilis plays a much more important part than rickets.

The skull is usually broad, high, and in its lateral portions protuberant, while in the middle of the forehead the surface is flat. This condition is brought about by the thickening of the

frontal tuberosities. The same condition occurs over the parietal tuberosities, though, as a rule, the tuberosities of the frontal bone are the most markedly thickened. In the region of the coronal suture there is often a depression, dividing the skull apparently into an anterior portion and a larger posterior portion of the skull. The parietal bones project outward and appear to be overhanging. The four tuberosities are not all enlarged in the same degree, and where there is considerable irregularity there may be an apparent asymmetry of the skull. For instance, on one side the tuberosity looks forward and externally, while on the other side the tuberosity is flat, thus producing a very marked asymmetry. At another time the tuberosities of the frontal bone are very markedly thickened and very prominent, while those of the parietal bone are almost unaffected. In such a case the increased prominence of the frontal portion of the skull causes the appearance of marked enlargement in the anterior portion. Sometimes the borders of the sutures are swollen and give the impression of being protuberant. The anterior fontanel closes normally at about the fifteenth month; in rickets the fontanel sometimes does not close until the end of the second year or even later. The two posterior lateral fontanels may remain open for a long time and the sutures may close abnormally late. In rachitic children the sutures may remain open until the second year. The bones that form the boundary of the fontanel are frequently soft and compressible in marked cases of rickets. Softness and compressibility of the skull bones occur in various parts of the skull and is the condition which is called craniotabes. Craniotabes occurs very frequently at the margin of the fontanel and in the posterior portion of the occiput and sometimes over the parietal bones. The areas of softening vary in size from a kernel of corn to a five-cent piece, or the areas may become confluent and form very large softened areas of bone; or, indeed, in the very severe cases the entire occiput seems so soft and compressible that all normal bony resistance is lost. The consistency of these soft areas varies—sometimes it is like parchment and crackles; at other times it is much softer, and resembles

the sensation which one obtains when palpating a soft-boiled egg with the shell removed or a distended ice-bag.

Rickets of the Spine.—The spinal column in florid rickets is very easily bent and, indeed, if the child be placed toward one side or the other, curvatures in the bony structure may be observed, depending upon the position, and thus a temporary kyphosis or scoliosis or lordosis may be produced at will. The bending does not occur in a single vertebra, but a large number of bone elements are involved at the same time. Rachitic kyphosis is probably the most common curvature. It occurs most often in the lumbar and in the dorsal region. Permanent rachitic deformity in these regions may occur. On the other hand, permanent rachitic deformities of the cervical vetrebra do not occur. When rachitic lordosis occurs it is located in the lower dorsal and in the lumbar vertebra, and usually compensates a rachitic malposition of the pelvis, and, indeed, rachitic changes of the spinal column are frequently associated with deformities of the pelvis and the lower extremities. It may be suggested here, too, that asymmetric rachitic curvatures in the legs with static disturbances may lead secondarily to scoliosis.

Changes in the Thorax.—The most frequent change in the thorax is the so-called rachitic rosary, and this occurs at the junction of the bony with the cartilaginous portion of the rib. This rosary is often visible to the naked eye, and appears like knobs or buttons arranged in a row on either side of the thorax and from above downward. The rosary of the eleventh rib is usually observed in the midaxillary line. In obese children the rosary, as a rule, is not visible, but must be palpated. Occasionally one notes that there is some irregularity in the size of the individual knobs or buttons. Over one rib the rosary may be absent, and, indeed, the individual knobs may vary very much in size. It is true that sometimes where there is great lateral rachitic depression of the thorax the rosary may be absent. It is not infrequent that flattening of the lateral portion of the thorax is observed in rickets. Sometimes the sternum is unduly prominent, and in those cases where there is considerable kyphosis the sternum may be convex and project forward. The thoracic

deformities in rickets are usually symmetric, though sometimes there is more marked change on one side than the other. As a general rule, a rachitic thorax is small in its upper two-thirds, particularly if the circumference be compared with the head and the abdomen. The so-called "chicken-breast" occurs not infrequently as the direct result of rachitic deformity. The rachitic deformities of the thorax are easily accounted for when one considers the pathologic softening of all the bony structures, the natural loss of elasticity which results, and possibly the diminution of intrathoracic pressure and the disproportion between the latter and the atmospheric pressure. In addition to this, the distention of the abdomen so common in rachitis increases the circumference of the lower thoracic aperture and possibly tends in this way to diminish the pressure within the thorax. The lateral depressions of the thorax frequently correspond with the position of the arms, and are thought to be caused by pressure of the arms in this position. The rachitic deformities of the thorax may be further complicated by fractures or by traumatic curvatures of the ribs.

The clavicles usually increase in thickness, and the normal grotesque curvature is increased and becomes pathologic. Not infrequently infractions occur in the clavicle; very often callous formation occurs at the site of the infraction, giving rise to marked deformity.

It is worthy of note, too, that deformities of the clavicle are frequently symmetric, both sides being equally involved. The scapula may be thickened or the spine of the scapula may project outward.

The humerus is only rarely curved. The enlargement of the epiphysis is difficult to make out at the shoulder-joints. At the elbow-joints the enlargement is more easily felt, and in thin children is visible. The humerus may show infractions at times, about the middle of the shaft. The arm may be shortened in some cases. This is as a result of arrested growth in the long direction. In the forearm the enlargement of the epiphysis is one of the most constant symptoms. The epiphysis of the ulna and the radius are increased in size and thickened. The di-

aphysis of the bones of the forearm not infrequently show curvatures, and in cases of severe rickets marked curvature may be noted just above the epiphysis, and, indeed, infractions of the bones of the forearms may occur after convulsions or after moderate trauma. In addition to this, the radius and ulna may be curved in a spiral way, and this sometimes leads to deformities of the hand, causing a pronation which may be more or less prominent. It may be noted, too, that while the upper extremities are frequently involved in rachitic processes, the changes are not so severe and not so constant as they are in the lower extremities.

The changes in the pelvic bones are manifested principally by the approximation of the promontorium of the sacrum and the symphysis pubis, leading to a flattening and an increase in the transverse diameter.

The Order in Which the Lesions of Rickets Appear.—It may be stated that the bones are affected in the order in which they may develop functionally, and that when bones are brought into use rickets becomes manifest. During the first month of life the head and the thorax grow most rapidly, consequently we find rickets appearing earlier in these two localities, and consequently the rosary and craniotabes may be considered the earliest symptoms. Then the spine is noted to show changes early and it tends to become kyphotic. This is due to the fact that the child may be continuously on the arm of mother or nurse, and because of the weak condition of the muscles is unable to support the softened bony column; consequently kyphosis is observed. Sometimes the craniotabes becomes marked in the second year. In those cases where rickets occurs at the close or the end of the first year craniotabes does not occur, and the changes in the bony thorax are less marked, if they occur at all. Most prominent are the rachitic changes which occur in the extremities, and this is particularly true in those cases that occur after the second year of life. In those cases where rickets has developed as late as the second year, kyphosis and scoliosis of high degree and enlargement of the frontal and parietal tuberosities of the skull are very marked. In those cases where rickets begins some time during the first half-year and continues in a florid state until the second

year, it is noted that the craniotabes disappears some time before the first year, while the rachitic process in other respects may continue until the second or the third year. If we stop for a moment to consider that period in the course of rickets which indicates an improvement or a healing of the rachitic process, we note that the first change toward recovery is the disappearance of the softening of the thorax and also of the cranial bones. In addition to this, the abnormal size of the fontanel tends to grow less, the periosteal thickening diminishes, the swelling of the epiphysis and the curvatures of the various bones become less marked and tend to improve; and many of these conditions occur without very much attention to treatment. It is remarkable how sometimes severe deformities as bow-legs or knock-knees seem to make spontaneous recoveries. Very often the enlargements over the tuberosities, over the frontal and parietal bones almost disappear, leaving only slight evidence of the pre-existing disease. Of course, it is not to be denied that where severe deformities have occurred residual deformities of one form or another will remain during the entire life of the individual.

Other Organs and Functions.—Sometimes children who are breast fed develop a mild degree of rickets, and in these patients it is frequently noted that they fail to gain in weight, and that the normal tone of their tissues is inferior. Sometimes these children seem fat, though they are below normal in weight and the tissue soft and relaxed. In florid rickets, particularly, children are pale and, indeed, in some cases they have a grayish-yellow color or some have a dirty cachectic color. In some of these cases the skin is atrophic, or it may be thin, relaxed, and may fall into folds.

The examination of the blood in rachitic children is not entirely characteristic. The red cells may be scarcely diminished, though this is not always so. There may be a diminution in the number and a change in the form of the red cells. The hemoglobin is usually diminished; a leukocytosis may occur in some cases.

There has been a great deal of discussion as to the bodily temperature in relation to rachitis. It has been pretty generally conceded that uncomplicated rickets is perfectly afebrile; that when fever occurred, some complication, such as bronchitis or

gastro-intestinal infection or food disturbance, was responsible. It is true, of course, that fever may be very obscure in young infants, and that it is sometimes difficult to explain the occurrence of fever in florid rickets, especially if no complication or concomitant lesion is present.

The Relation of the Psychic State to Rickets.—The mental development is, as a rule, not influenced by rickets. These children learn to talk at the usual time, and, indeed, rachitic children are frequently very bright. In the severest cases, however, some interference with intellectual development seems to occur, and this without any cerebral lesion. Undoubtedly in the severe cases, where there are marked nutritional disturbances, the explanation lies close at hand. Perhaps a cause for delayed intellectual progress is to be found in the fact that many of the rachitic children learn to walk late, that they are alone much, that they have little concourse with other children, and consequently miss the association with other children which stimulates mental action. It is true, too, that many rachitic children are fretful, restless, easily frightened, and it is thought that a great deal of the restlessness and mental irritation from which these patients suffer is due to pain. We have already mentioned the fact that in florid rickets, particularly, the bones are painful if pressed, but in some cases the muscles also seem to partake of this sensitiveness. It is often noted that if a rachitic child be grasped under the arms and lifted up somewhat forcibly it begins to cry as though in severe distress. It has been said that rachitic children have diminution in the taste sensation. The musculature of rachitic children is, as has been so frequently referred to, relaxed and is frequently atrophic. This is particularly true of the lower extremities. In some of the severer cases the muscular tissue may atrophy extraordinarily. The electric excitability in the case of simple rickets usually remains normal. The muscular atrophy is probably due to disuse. Rachitic children do not desire to move; they often lie quietly on one side or on the back, and this undoubtedly has something to do with the relaxation of the muscles as well as that of the ligaments.

There are some changes in the topography produced by rickets

which are of interest from the standpoint of physical diagnosis. The supra- and infraclavicular fossæ are deepened on account of the curvature of the clavicles. The percussion note is less resonant over the bulging portions; on the anterior surface the percussion note is sometimes less clear and loud over the left side than on the right side and over the sternum.

On examining the back, one finds differences in percussion on one side or the other, even when the child is held in good position. This may be due to asymmetric rachitic deformity of the ribs or possibly to rachitic scoliosis. Auscultation of the thorax sometimes shows in rachitic children an exaggerated vesicular note, which has been referred to as a pseudobronchial sound. It occurs loudest where the percussion note is clearest and most resonant, and is not to be heard over the areas where dulness on percussion is obtained. In the diagnosis of pulmonary disease in a rachitic baby auscultation seems to have more importance than percussion, and one should not depend for localization of pulmonary lesions upon refinements of percussion because of the thickened, bony deformities, and malpositions which have already been referred to. If it is kyphosis, the position of the heart may be changed; the diaphragm and liver are subject to change of position, and consequently changes in physical signs occur. It may be noted, too, that in recent years pathologic changes in the heart muscle have been described as occurring in rickets. We know that the pulse is frequently increased; the digestive function may remain undisturbed; the appetite in some cases is diminished, though sometimes increased, and rachitic children frequently desire large quantities of bread, potatoes, and sweets. Frequently rachitic children have great thirst. The abdomen is frequently distended; as a rule, soft and not sensitive. The evacuations are varied, frequently constipated, though constipation may alternate with diarrhea. Frequently the stools are only slightly colored. In severely sick rachitic children, with markedly distended abdomen, feces of peculiar soapy consistency occur—light in color and very offensive odor.

The liver is frequently enlarged. It is true that this enlargement may be more apparent than real. On account of

descent of the diaphragm in thoracic rachitis the liver may be pushed downward.

It is commonly thought that the spleen is enlarged, though most authors are agreed that enlargement of the spleen occurs only where there is considerable anemia, and what has been said of the liver applies equally to the spleen—that it may be occasionally palpated because of the low position of the diaphragm.

In addition to the enlargement of the spleen in rickets we frequently observe swollen cervical, axillary, or inguinal lymph-glands. As a rule these children have some other disease than the rickets. Tuberculosis, diseases of the skin, such as furuncles and eczema, or gastro-intestinal diseases, may serve as focal points of inflammation, resulting in enlargement of the lymph-nodes.

Duration and Course of the Disease.—In the large majority of cases the first symptoms of rickets appear some time after the third month of life. From this time on the disease may appear until the second year. After the beginning of the second year the number of cases of fresh rickets are very few—that is, it is exceptional to see rickets develop after the second year. The active rickets may last several months, sometimes a year, occasionally two years or more. Rickets begins gradually, runs a chronic course; at times one notices periods in which the disease seems to have come to a close or a pause and then relapse follows.

"Acute rickets" is a misnomer. This term was formerly applied to scurvy, and the older descriptions which appear in the literature under this appellation were classic cases of Barlow's disease. When the florid rickets shows a tendency to improve the patient is less sensitive to pain, becomes more cheerful, and the older children may make attempts to stand or to walk; the color of the skin improves, and the softeness of the occiput and ribs tends to disappear. The teeth begin to erupt, the fontanel begins to undergo its normal evolution and diminishes in size, whereas it had been open and possibly stationary. Many times, too, when improvements occur these children show increase in growth, and eventually the deformities tend to improve.

Complications.—Children do not die from rickets *per se;* nevertheless many children suffering from rickets die from other diseases. We must recognize, too, that rickets predisposes children to certain diseases. There can be no doubt that on account of the relaxation of muscular tissue, especially of the abdominal wall, rickets predisposes to umbilical hernia. Possibly the same applies to inguinal hernia, though inguinal hernia is far less frequent than the umbilical variety. In point of importance and severity, the complications on the part of the respiratory tract are by far the most severe. Indeed, in cases of severe thoracic rickets pulmonary complications are likely to occur. The first evidence is the occurrence of bronchitis. One hears some râles over the posterior portion of the thorax, some dyspnea or inspiratory retraction is present. While it is true that such a bronchitis may make a spontaneous recovery in a few days, one always dreads possible results. One knows that frequently the course of such a bronchitis is protracted, lasts for weeks, and even after recovery seems to have occurred relapses appear; and then there is always the danger that the bronchitis will descend into the finer bronchi and constitute a capillary bronchitis or bronchopneumonia. The prognosis of this disease in rickets is always grave. We know, too, that pneumonia protracts in the rachitic child, the resolution is delayed, dyspnea, cyanosis, and fever may last for weeks, exhaustion continues, and prostration becomes extreme.

Next in importance to the respiratory complications are those on the part of the digestive system. Indeed, the gastro-intestinal disorders are frequently the cause of death in rachitic children in both hospital and private practice. The disturbances may be either infectious in nature or they may partake of food disturbances, giving rise to marasmus and frequently resulting fatally. It has been suggested that in certain cases dilatation of the stomach occurs as a complication of rickets. It is doubtful, however, whether there is sufficient evidence at hand to substantiate this opinion or, at least, to confirm the diagnosis in individual cases.

The nervous manifestations of rachitis are considered as a

separate disease and in a special chapter. Spasmophilia or tetany include the various nervous phenomena, such as laryngismus stridulus, increased electric excitability, and certain forms of infantile eclampsia. This subject will not be considered at this time. At another hour I shall consider in greater detail the subject matter of spasmophilic diathesis, including all the subtopics.

The **diagnosis** of rickets should offer little difficulty if one is awake to the general symptoms during the early course of the disease, disturbances of growth, and the manifestations on the part of the skeletal system. In young babies one should carefully palpate the skull to note the presence of craniotabes, the firmness or compressibility of the borders of the sutures and of the large fontanel. One should, of course, not mistake a congenital defect in the bones of the skull or hydrocephalus for craniotabes. Wherever there is delayed dentition, where the child learns to walk or stand late, rickets should be suspected and carefully searched for. In newborn babies there is sometimes a defective ossification of the bones of the skull which may suggest a craniotabes. It is difficult to differentiate this condition from genuine rachitic craniotabes. Attention has already been called to the fact that rickets occurs most commonly after the third month, and that craniotabes frequently appears during the first half of the year. It is true that craniotabes may be present shortly after birth, and may represent a condition of fetal or congenital rickets. In such cases, however, rachitic lesions will be present in other parts of the body. Atony of muscles, the presence of the rachitic rosary, assist in the early diagnosis.

So far as differential diagnosis is concerned, it is sometimes necessary to differentiate infantile myxedema from rickets. Indeed, many infants suffering from myxedema or cretinism are treated for years as cases of rickets. It is true, too, that babies with myxedema and those with rickets show many symptoms in common; for instance, in both may be found increase in the size of the costal end of the rib, thickening of the epiphysis, the eruption of teeth in both cases is delayed, closure of the fontanel is retarded, learning to walk and to stand are delayed. In myxedema the pulse is slow and the extremities are cool. In myxedema, it

must be remembered, there is a very marked arrest in the growth in the long axis of the body; there is retardation of psychic development. The baby with cretinism shows a characteristic physiognomy; the skin is dry, the hair is coarse, the tongue is thick and heavy. Most rachitic children perspire profusely; the myxedematous children perspire scarcely at all, as has already been pointed out; the skin in rachitic children is soft, in myxedema it is dry and sometimes thickened. In rachitic children the hair is thin and soft; in myxedematous children it is thick, coarse, and brittle. Myxedematous children have a poor appetite, and are very obstinately constipated to such a degree as rarely occurs in rachitic children.

Barlow's disease and infantile scurvy sometimes require differentiation. It is true that the two diseases are associated. A rachitic child often becomes scorbutic. In both diseases there is great sensitiveness if pressure be made over the bones. In Barlow's disease subperiosteal hemorrhages cause apparent swelling of the bone, especially over the diaphysis. One has only to recall that in Barlow's disease the gums are swollen, spongy, and bleed easily, hemorrhages occur into the eyelids and into the orbit. In rickets there is no tendency to hemorrhage, no disturbance of the gums, such as have already been alluded to.

Sometimes hereditary syphilis may come into consideration in the diagnosis of rickets. In both diseases the teeth erupt late, the psuedosyphilitic paralysis of Parrott, which I have already referred to at another hour, may sometimes be thought to be rachitic. In rickets the entire epiphysis is enlarged; in pseudoparalysis the margin of the epiphysis is involved; in syphilis the swelling is exquisitely painful, whereas in rickets the enlarged epiphysis is only moderately painful, if at all. In the pseudoparalysis, luetic in nature, the loss of motion is quite complete, whereas in rickets there is only moderate loss of motion.

The *x*-ray examination may be of great use in differentiating syphilis from rickets. The rachitic epiphysis shows a broad band at the junction of the epiphysis with the diaphysis, regular in outline, whereas the *x*-ray of the luetic child shows an undulated, serrated line at the junction of the epiphysis with the diaphysis.

Prophylaxis and Treatment.—Proper feeding is undoubtedly the most important factor in prophylaxis. There can be no doubt that a well-regulated breast feeding is the most efficacious plan in the prevention of rickets. It has been maintained, though it has not been proved, that long-continued lactation tends to cause the disease. Overfeeding, even of breast milk, is undesirable, and likely to lead to chronic digestive disturbances. It has long been taught that the early supplemental feeding of starches and animal broths is contra-indicated. There is, however, strong clinical evidence that the judicious introduction of these supplemental foods into the diet at the seventh or eighth month may prevent the development of rachitis. There can be no doubt that rickets is less frequent and less severe in its manifestations, and more readily cured if present, if the patient can be kept out-of-doors, receive an abundant supply of fresh air and sunlight. There is no more efficient plan of treatment than to advise a visit to the country or the mountains or the seashore. Fresh air and sunlight are sometimes more valuable than medicine in the treatment of this disorder.

On account of the softness of the bony structures during the first stage of this disease children should not be permitted to occupy the same position in bed for too long a time, nor should they be allowed to lie continuously on their backs with the occiput pressed on the pillow, because a long continuance of the same position in bed causes pressure on the soft bony parts and is likely to lead to deformity and indentation, particularly of the occiput.

It is also a matter of experience that rachitic infants who are held or carried continuously in the same position are likely to suffer from spinal curvatures. The position of the infant should be changed frequently from one arm to another, and the back and head should be well supported. For the same reason, rachitic babies should not be allowed to walk or stand too soon, on account of the danger of producing curvature of the lower limbs.

The bathing of infants suffering from rickets is of therapeutic value. Salt baths are beneficial in those children who perspire profusely, and frequently children are sent to saline bath resorts,

where they receive benefit from the out-of-door life and, possibly, the baths themselves.

Dietetic Treatment.—It is difficult at this time to pronounce the final word on the dietetic treatment, though in a general way it may be said:

(1) The food should be adapted to the ability of the infant to digest without giving rise to intestinal putrefaction or fermentation. (2) Overfeeding should be avoided and, as a rule, five meals, at the most six, should be given daily. Animal broths, beef juice, orange juice, and vegetable broths should be given early, in some instances as soon as the sixth month. In severe cases of refractory florid rickets the animal broths, particularly veal broth, may be given in early infancy.

The interval between feedings should be at least three hours. In many instances a four-hour interval is still better. It seems plausible that long rests between feedings favor more complete digestion and assimilation.

Experiments have not proved that sterilized milk is capable of producing rickets. There are some foremost clinicians who believe that boiled milk is capable of producing the disease. Even in the latter instances the evidence is insufficient. Nevertheless, one may begin early, say the fourth or fifth month, by fortifying the milk feeding with orange juice, beef juice and, in some instances, animal and vegetable broths. Clean, fresh unboiled milk properly diluted is preferred by the majority of physicians. Clinicians are almost unanimous in condemning as rickets producers the long-continued use of condensed milk, dried milk, and some of the proprietary foods which are diluted with water and ready to serve.

Medicinal Treatment.—Chickens, rabbits, and other animals to whom phosphorus in an elementary form was administered showed a sclerosis of the bony tissue. Following this observation, Wegner administered phosphorus to children who were suffering from rickets. Kassowitz, after many years of clinical and histologic study, has insisted that phosphorus in minute doses is the most valuable drug in the treatment of rickets. Jacobi, who recognizes the virtue of phosphorus, or phosphorated oil, has

pointed out that phosphates are of practically no value in the treatment of rickets.

It has been shown that phosphorus combined with cod-liver oil favors the retention of calcium. Whether this indicates that it favors the activities of calcium metabolism is not known. The empiric evidence in favor of the use of phosphorus, as phosphorated oil, or its combination with cod-liver oil, more than justifies its employment. The various preparations of calcium, whether in the form of lime-water or phosphates or lactophosphates, are certainly of no value. Cod-liver oil alone is of doubtful value.

In the early cases, or in florid rickets, phosphorus treatment is especially indicated. The oleum phosphoratum, each minim of which represents $\frac{1}{100}$ grain, may be given in $\frac{1}{2}$- to 1-drop doses three times daily, diluted in cod-liver oil or a small portion of milk.

Phosphorus treatment must be continued for a long time in order to produce results. Frequently, under this treatment, walking is hastened, the teeth appear earlier, and the anemia becomes less. The treatment, too, has a favorable effect on the spasms of the larynx, the head sweating, and the convulsions.

The treatment by organotherapy, such as the use of the thyroid, the thymus, and the suprarenal gland substances, has been used only to be abandoned, since these remedies have proved themselves absolutely worthless in the treatment of this disease.

Where enlargement of the glands occurs, syrup of iron iodid, cod-liver oil, syrup of hydriodic acid, or the iodids of potassium or sodium may be useful.

Massage applied intelligently exerts a favorable influence on affected muscles which are relaxed and inactive. The massage must be performed by one who has considerable experience, and the overzealous operator who is accustomed to treat adults must modify her operations and develop a special technic of a gentle character suitable in the treatment of young children.

Deformities.—The rachitic deformities occurring in bones require special consideration aside from the medicinal, dietetic, and hygienic treatment which have already been referred to. A beginning kyphosis or scoliosis may be prevented, especially if

the muscles of the back are relaxed, by not holding the child continuously in one position. It is better that such patients be changed from one arm to another, as has already been stated, and that the bed be so arranged that the mattress remains smooth without depressions or elevations. In the same way, the child should not be allowed to lie continuously on one side. If the extremities show deformities during the early stages of rickets, while the bones are still soft, light splints may be applied. As a matter of fact, active orthopedic treatment is not called for in the early cases. The consensus of opinion among all surgeons at present is that, as the muscles acquire normal tonicity and undergo permanent development, the bony deformities tend to correct themselves spontaneously, even as late as the fourth or fifth year.

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